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AN APPRAISAL OF PRESENT SCIENTIFIC INFORMATION CONCERNING PERIODONTAL DISEASE

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ST. LOUIS, MO.—SEPT. 8-11

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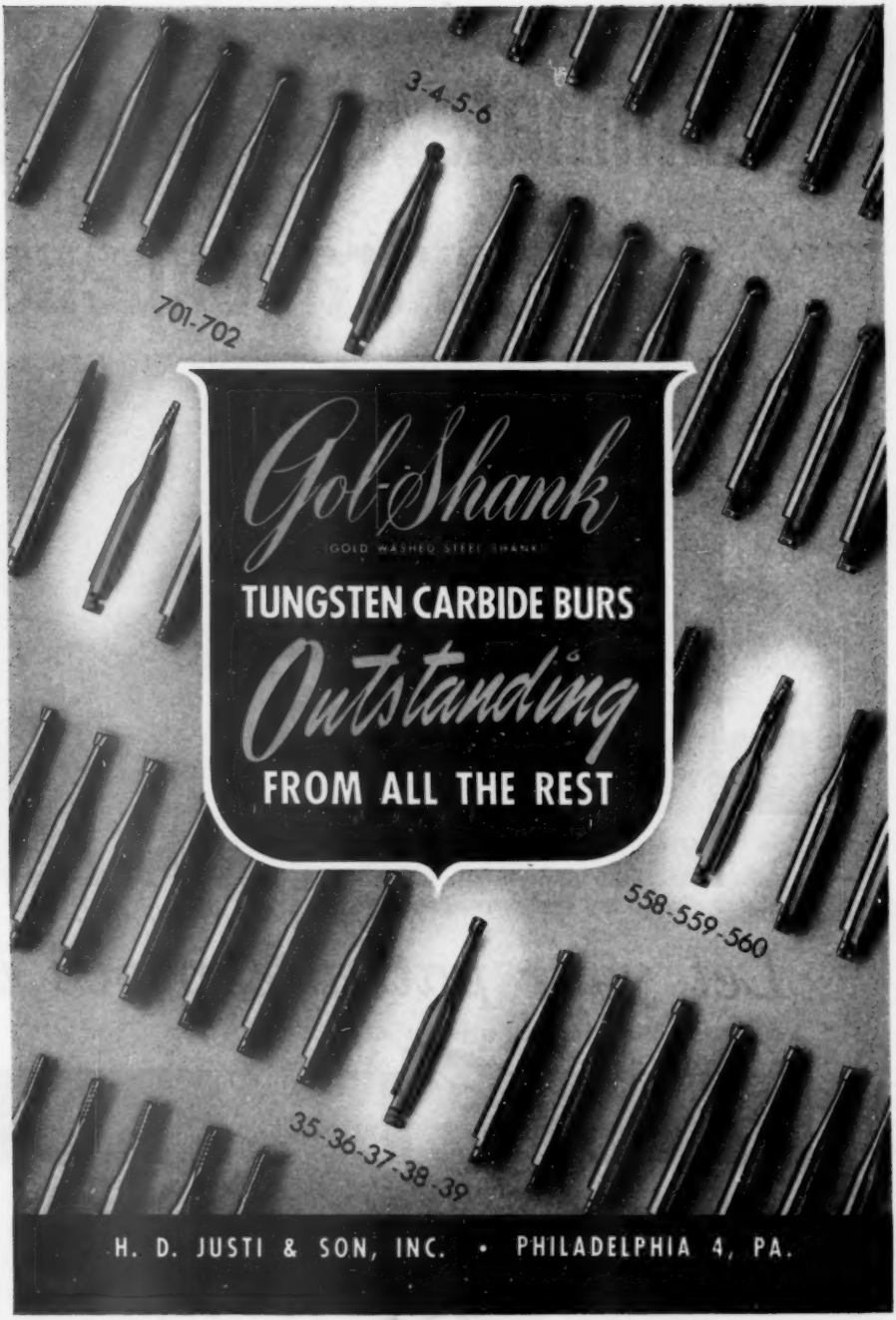

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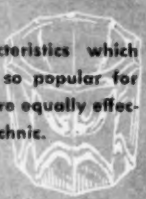

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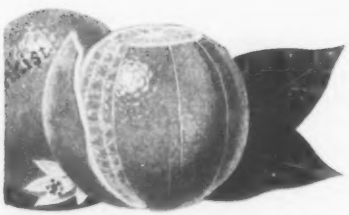

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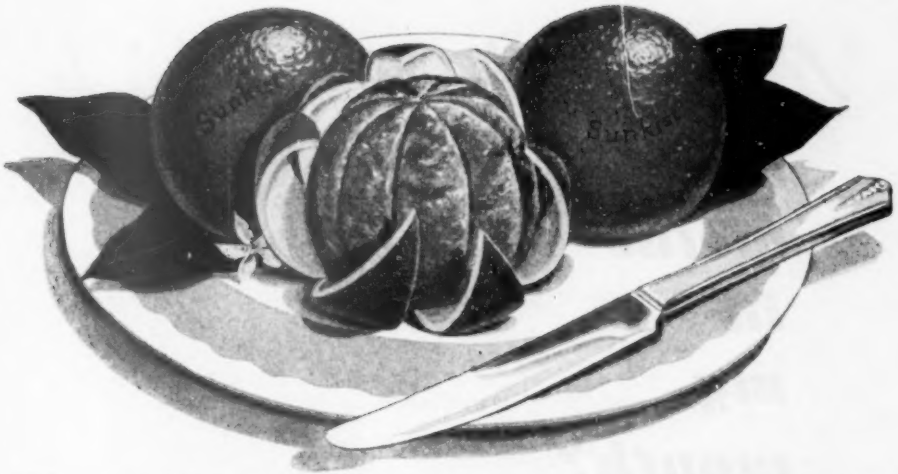

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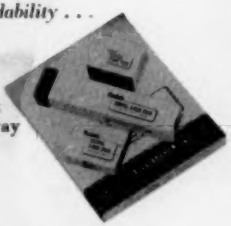

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Periodontal disease

ETIOLOGY, PATHOLOGY, PREVENTION AND TREATMENT

JUNE 1952

THE Journal OF THE AMERICAN DENTAL ASSOCIATION

VOLUME 44 • NUMBER 6

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PAPERS PRESENTED AND CONCLUSIONS DRAWN AT AN INSERVICE TRAINING COURSE CONDUCTED AT THE UNIVERSITY OF MICHIGAN SCHOOL OF PUBLIC HEALTH AND SCHOOL OF DENTISTRY, ANN ARBOR, MICHIGAN SEPTEMBER 10-15, 1951

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HENRY F. VAUGHAN, *dean, School of Public Health*

Classification and terminology for 1951 periodontal workshop

Donald A. Kerr, D.D.S., M.S., Ann Arbor, Mich.

Recognizing that a universally acceptable system of nomenclature is impossible it is necessary, nevertheless, to attempt to standardize and unify terminology as much as possible.

It is not the purpose in this workshop to select a universally acceptable terminology or classification but it is necessary to adopt a terminology and classification which will permit uniformity in the function and reporting of the results of this workshop. Therefore, a classification and terminology have been selected in order to prevent confusion in the discussion during the workshop and not for the purpose of establishing a new and universally acceptable classification and terminology.

The Academy of Periodontology has attempted to establish a universally acceptable nomenclature and has appointed a committee on terminology and nomenclature. The results, however, have been unsatisfactory because the committee membership changes yearly as does the nomenclature as each group attempts to incorporate their personal preference or prejudice into the committee report. Because of the constantly changing character of the nomenclature accepted by the Academy, it seemed unwise to adopt the suggestions of any one committee for use in this workshop. For this reason the classifications and terminology listed herein have been selected arbitrarily for use in the workshop.

TERMINOLOGY

The following anatomical, histological and pathological terms have been selected and defined as they are to be used in this workshop.

Alveolar Bone • That portion of the alveolar process which forms the wall of the alveolus and into which the periodontal fibers are attached. Roentgenographically this is designated lamina dura.

Alveolar Crest • The thin margin of bone forming the coronal edge of the alveolus. It is continuous with the alveolar process.

Alveolar Process • All of the cortical and cancellous portions of the maxillary bones except the alveolar bone.

Bruxism • Gnashing or grinding of the teeth in other than normal function.

Calculus • Calcareous deposit on the teeth resulting from the deposition of inorganic salts of the saliva in an organic matrix. This deposit may be supragingival or subgingival.

Epithelial Attachment • The portion of the epithelium which is attached to the tooth surface from the base of the gingival sulcus for a variable distance apically. On the crown

Associate professor of dentistry, head of departments of periodontia and oral pathology, University of Michigan School of Dentistry, Ann Arbor, Michigan.

this is the reduced enamel epithelium and on the root it is the result of proliferation of the epithelium along the cementum.

Gingiva • The gum—the fibrous tissue and overlying mucosa which covers the alveolar process of the upper and lower jaws and surrounds the necks of the teeth.

Gingiva, Attached or Alveolar • The portion of the gingiva attached to the cementum and alveolar process. It extends from the free gingiva to the mucogingival junction.

Gingiva, Marginal or Free • The thin border of the gingiva which rests against the tooth cervix. Its inner surface lines the gingival sulcus and normally is in contact with the enamel in young persons or the cementum in the aged.

Gingival Sulcus • The shallow groove which develops between the gingiva and the surface of the tooth around its circumference. It is bounded by the surface of the tooth on one side, and by the gingiva on the other. The bottom of the sulcus is the epithelial attachment.

Oral Hygiene • The establishment and maintenance of a clean mouth free of calculus, stains, and soft debris.

Oral Prophylaxis • Removal of supragingival and subgingival accretions from the teeth by the use of scalers and polishing instruments as well as the removal of overhanging margins of restorations.

Periodontium • The gingiva, alveolar bone, cementum and periodontal membrane.

Periodontal Disease • Diseases of the periodontium. One or more of the components of the periodontium may be involved.

Pocket, Gingival • Increase in depth of the gingival sulcus associated with gingival inflammation but without apical proliferation of the epithelial attachment.

Pocket, Intrabony or Intraalveolar • A pathological pocket, the base of which is apical to the alveolar crest.

Pocket, Periodontal • Pathologic deepening of the gingival sulcus produced by destruction of supporting tissues and apical proliferation of the epithelial attachment.

Subgingival Curettage • Removal of debris, calculus, and epithelium from a periodontal pocket followed by filing and smoothing the root surfaces.

Systemic Disease • A disease affecting the body as a whole.

CLASSIFICATION OF PERIODONTAL DISEASE

The following classification has been selected for this workshop. It is believed to be all inclusive and easily workable. Several other classifications are presented for the purpose of correlation, to aid in the interpretation and integration of terms used by various authors in describing types of periodontal disease.

Gingivitis •

- Simple—Acute or chronic inflammation due to poor oral hygiene.
- Infective
  - Coccic—Due to staphylococci, streptococci gonococcus, pneumococci, and so forth.
  - Vincent's (Fusospirochetal, ulceromembranous)—Associated with action of fusiform bacilli and Vincent's spirochete.
- Herpetic gingivostomatitis—Acute process due to initial invasion of herpetic simplex virus.
- Hyperplastic
  - Simple—Proliferative reaction to chronic inflammation.
  - Hereditary gingival fibromatosis—Progressive proliferative process.
  - Dilantin—Progressive proliferative process caused by Dilantin.
  - Leukemic—Increase in bulk due to infiltration by neoplastic blood cells.
- Hormonal
  - Pubertal—Hormonal stimulation at age of puberty.
  - Pregnancy—Hormonal stimulation during pregnancy.
  - Chronic desquamative—Altered premenopausal hormonal stimulation.
  - Chronic atrophic senile—Postmenopausal hypogonadal function.
- Atrophic
  - Physiological—Gingival recession or passive eruption.
  - Pathological—Gingival recession excessive for age.

Periodontitis • Sequelae of untreated gingivitis with extension of inflammation to deeper periodontal structures, actual pocket formation, and loss of alveolar bone.

Periodontosis • Degenerative changes in periodontal membrane, loss of alveolar bone, spon-

taneous wandering, late pocket formation. Inflammation absent until after pocket formation.

Traumatism • Gingival recession, alterations in width of periodontal space. Uneven thickening of lamina dura, looseness of teeth, loss of alveolar bone. Etiology: excessive function.

OTHER CLASSIFICATIONS

In Gottlieb's classification[1] gingivitis is divided into three types: Vincent's infection which is the process associated with fusospirochetal infection; Schmutz pyorrhea which is produced by poor oral hygiene, and hyperplastic gingivitis which includes simple hyperplastic gingivitis, hereditary gingival fibromatosis, Dilantin gingivitis, leukemic gingivitis and hormonal gingivitis.

Paradental pyorrhea is periodontitis, and diffuse alveolar atrophy is periodontosis. In 1946 Gottlieb[2] suggested the term cementopathia to describe the process of periodontosis. However, later he suggested that the term cementopathia should be used to designate periodontosis and periodontitis.

Orban has suggested two classifications. In his original classification[3] gingivitis was divided into local and systemic types. The local gingivitis included simple, infective Vincent's, herpetic and mild hyperplastic. The systemic type included hereditary, Dilantin, leukemic and hormonal gingivitis.

In this classification hyperplasia is a specific category containing the systemic forms of gingivitis which are characterized by increase in bulk of the gingiva.

In 1950 Orban[4] proposed a new classification in which the periodontal lesions were divided into three types—inflammatory, regressive and productive. The gingival processes characterized by inflammation are termed gingivitis. Acute and chronic gingivitis is of the simple type. Vincent's and herpetic processes would be classified under ulcerative.

The term, "gingivosis," coined by Schour and Massler, is placed under the forms of periodontal disease characterized by degeneration (regressive group). Gingivosis indicates the process previously called chronic desquamative gingivitis. This condition and atrophy are retrogressive changes and are included in this category.

Those forms of hyperplastic gingivitis having a systemic etiology are classified as "productive."

The Box classification[5] is divided into gingivitis, rarefying pericementosis fibrosa and periodontitis. Gingivitis is divided into acute and chronic, with ulcerative, nonulcerative and hyperplastic under each type. This would include all of the forms of gingivitis included in the workshop classification. Rarefying pericementosis fibrosa is the term used to designate traumatism. Periodontitis is divided into simplex type, which is the same as the workshop classification, and periodontitis complex which represents periodontosis. The degree of involvement and the pathological process are indicated by subgroups under the two forms of periodontitis.

In Goldman's classification[6] functional gingivitis includes a form of atrophy as well as the changes of occlusal traumatism. Goldman's designation of syndrome is what Orban calls periodontitis complex and, as classified for this workshop, is periodontosis in which pocket formation has occurred.

In Schour's classification[7] gingivitis is

1. Gottlieb, B., and Orban, B. Zahnfleischentzündung und Zahnlockerung, ed. 2. Berlin, Berlinische Verlagsanstalt, 1936.

2. Gottlieb, B. The new concept of periodontoclasia. J. Periodont. 17:7 (Jan.) 1946.

3. Orban, B. Classification and nomenclature of periodontal diseases. J. Periodont. 13:88 (July) 1942.

4. Orban, B. Periodontal diseases. Texas D.J. 68:429 (Oct.) 1950.

5. Box, H. K. Studies in periodontal pathology. Bulletin no. 7. Toronto, Canadian Dental Research Foundation, May, 1924.

6. Goldman, H. M. Periodontia, ed. 2. St. Louis, C. V. Mosby Co., 1949.

7. Schour, I. Current problems in terminology and classification of gingival and periodontal disease. J. Periodont. 22:10 (Jan.) 1951.

classified according to anatomic location regardless of the etiology or pathologic change. Periodontitis is divided into two types depending on severity. Gingivosis is used in Schour's classification to designate degenerative changes in the gingiva and is indicative of the same changes termed gingivosis by Orban. Traumatic periodontitis proper is the term used by Schour to indicate traumatism.

Numerous other classifications are available but these are the ones most frequently referred to in the literature. This brief correlation should serve to integrate them with the classification selected for this workshop.

Histology and physiology of the gingiva

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The gingiva is that part of the oral mucous membrane[1] which surrounds the teeth as part of the so-called masticatory mucosa[2] (Fig. 1). Two parts are distinguished in the gingiva—the free gingiva and the attached gingiva. The free gingiva extends from the bottom of the gingival sulcus to the coronal margin. The bottom of the sulcus often is marked on the surface of the gingiva by a fine groove which runs parallel to the margin, the free gingival groove. The free gingival groove is a line of demarcation between the mobile free gingiva and the firmly-anchored attached gingiva. It is an expression of functional differences between the free and the attached gingiva, a line of friction (Fig. 2), and histologically it is indicated by a heavy epithelial ridge (Fig. 3). The attached gingiva extends from the free gingival groove to the mucogingival junction; its stippled surface presents an appearance resembling that of an orange peel.[3] It is firmly anchored to the surface of the tooth and alveolar crest (Fig. 2) by coarse collagenous fiber bundles. The presence of these fiber bundles is responsible for the surface stippling of the gingiva (Fig. 4). The knowledge of this fact is important in the clinical appraisal of disappearance of stippling which may be caused by inflammation, edema or degeneration of connective tissue elements.[1, 4] The clinical differentiation of a free and an attached gingiva has a functional and structural background and is of diagnostic importance.

The surface of the epithelium of the gingiva (free, as well as attached) is keratinized.[5] The epithelium, toward its connective tissue side, shows regular ridge formation.[6] The epithelium of the alveolar mucosa is not keratinized and shows

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no ridge formation. The connective tissue of the alveolar mucosa is poor in collagenous fiber bundles but rich in elastic fibers, which are not present in the gingiva.[6]

The attachment of the gingiva to the surface of the tooth is of vital concern in this discussion. The fact that the epithelium of the oral mucous membrane is attached to the surface of the tooth, creates a biologic problem which was discussed by Gottlieb in 1921.[7] Gottlieb demonstrated that the attachment of the epithelium of the gingiva to the tooth is broad and cuff-like. This attachment first is limited to the enamel, later to enamel and cementum, and finally to the cementum only. Before Gottlieb's description of this epithelial attachment, it was generally believed that the epithelium of the gingiva was attached to the tooth by a few cells only—at the "gingival line," a landmark called the cemento-enamel junction.[8]

The discovery of the epithelial attachment not only changed the entire concept of periodontal diseases, but it has been the foundation of extensive and continuing research concerning the biology and pathology of the supporting tissues of the teeth.

The mode of attachment of the epithelium to the enamel is not yet a settled question. It is, however, reasonable to assume that there exists a cementing substance, which firmly and organically attaches the epithelium to the surface of the enamel. It must be remembered that the epithelial attachment to the enamel originates from the epithelium which produced the enamel. This epithelium, in turn, originates as an early development from the ectoderm of the primitive oral cavity. It goes through stages of proliferation and differentiation into enamel-producing epithelium. After fulfilling this function, it regresses to a stratified type of epithelium, similar in structure to the one from which it originally came. This so-called reduced enamel epithelium (reduced in size and function) remains in organic union with the enamel, probably through the medium of an organic, highly polymerized cementing substance.

In the progressive shifting of the epithelial attachment from the enamel to the cementum, two processes have to be considered. First, the separation of the epithelial attachment from the surface of the tooth at the bottom of the gingival sulcus. This is caused by the physiologic shedding of epithelial cells on the surface. The bottom of the gingival sulcus has to be considered the surface of the epithelial attachment. Because of the continuous degeneration of epithelial

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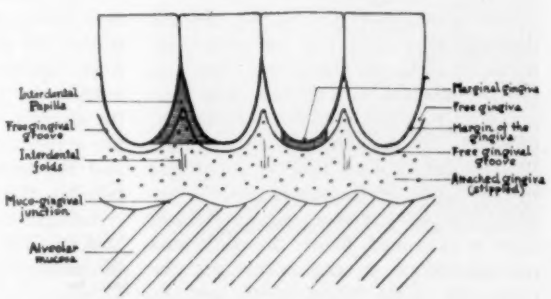

Fig. 1 • Diagrammatic representation of the gingiva in a frontal view

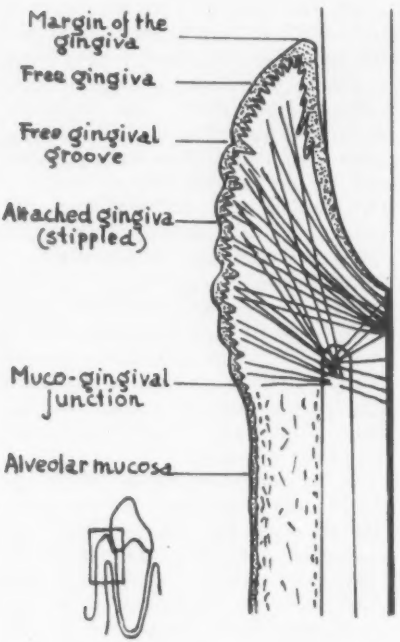

Fig. 2 • Diagrammatic representation of the gingiva in a labiolingual section

cells at the bottom of the gingival sulcus, the epithelial attachment is continuously separated from the surface of the tooth. This physiologic process is aggravated by the presence of microorganisms and by other local irritational or systemic factors.

The second process to be considered is the shifting of the apical end of the epithelial attachment from the cementoenamel junction to the cementum. The apical end of the epithelial attachment, originally at the cementoenamel junction, begins at some time to proliferate to the surface of the cementum. To explain this phenomenon, several factors must be examined. Before the epithelium can proliferate to the cementum, the connective tissue fibers which are anchored into the cementum have to degenerate. The first step in this process has to be the calcification of the cementum which has formed last, and the cessation of formation of new cementum. The second step is the degeneration of Sharpey's fibers in the cementum and the third step is proliferation of the epithelium. The modus operandi of this process is not yet established. There are several theoretic explanations, all of which may have merit. One factor should be kept in mind; the proliferation of the end of the epithelial attachment always takes place before the separation of the attachment at the bottom of the sulcus or pocket has reached the end of the epithelial attachment. There must be some regulatory mechanism which will bring about the degeneration of connective tissue, cessation of cementum formation and proliferation of the epithelium.

Toxins from the ever present inflammatory process close to the bottom of the gingival sulcus or from the gingival sulcus following a route along the intercellular spaces of the epithelium; desmolytic action of the epithelium; loss of vitality of the cementum; all have been theoretically taken into consideration without giving an all-around, satisfactory explanation. The action of several factors, at different occasions and times, might have to be considered.

Leaving the problem of the epithelial attachment and surface characteristics of the gingiva, the connective tissue elements in the gingiva will be discussed. In the attached gingiva, and to a certain extent, in the free gingiva, the connective tissue fibers appear in coarse bundles which anchor the gingiva to the tooth and to the alveolar crest. Fiber bundles run from the cementum to both the free gingiva and the attached gingiva. Strong fiber bundles also run from the bone to the cementum and radiate toward the free and attached gingiva. Some bundles on the periosteal side of the bone cross the alveolar crest to attach to the cementum,

Fig. 3 • The heavy epithelial ridge corresponding to the free gingival groove

as shown lately by Goldman;[9] other fiber bundles run directly from the bone to the attached gingiva.

The presence of such an elaborate fiber system has, of course, a functional purpose. It becomes evident that factors which lead to destruction of collagenous fiber bundles, or connective tissue in general, will affect the gingiva. Such factors might be of local or systemic nature. The term, "gingivosis," was coined by Schour and Massler[10] to indicate an obviously noninflammatory gingival disturbance in starving children. This term may be accepted on the basis that the condition is caused by enzyme action and that it produces a degeneration of collagenous fiber bundles and a depolymerization of the ground and cementing substance of the connective tissue.

The normal gingiva was studied recently for possible age changes and sex differences.[11] This study included 100 individuals with clinically normal gingiva between the ages of 4 and 80 years. A biopsy specimen was taken from each individual and the histologic features were studied by microscopic examination. Every specimen showed inflammatory cell accumulation in the free gingiva, close to the bottom of the sulcus. To eliminate the possible influence of inflammatory reaction, the study was confined to the attached gingiva.

The architectural pattern (histotype) of epithelium and connective tissue was studied and it was found that in the epithelium two types were present; one was with a medium suprapapillary width and medium long ridges and the other with a narrow suprapapillary width and long ridges. In the connective tissues, there were three types: (1) finely textured fibers (rich in cells); (2) irregularly textured fibers; (3) coarsely textured fibers (poor in cells). No sign of age changes was found in the epithelial cells; hornification was found in all ages. The connective tissue changed with ad-

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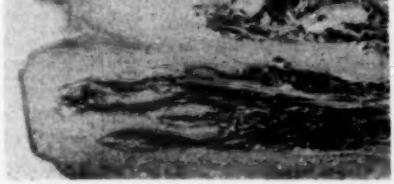

Fig. 4 • Fiber bundles which are responsible for the surface stippling of the gingiva

vancing age in that there was a decrease of cellular elements; the fibrous, intercellular substance increased and became coarser. Sex differences were observed only between the ages of 15 and 29 years; in females, the epithelium showed medium suprapapillary width and medium long ridges. The papillary connective tissue was finely textured. In some males, the epithelium showed a narrow suprapapillary width and long ridges. The papillary layer of the lamina propria was coarsely textured.

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Histology and physiology of the supporting structures

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The supporting structures of the teeth are cementum, periodontal membrane and bone. They are all connective tissue derivatives, modified for their particular use and function as a unit. The term "attachment apparatus" will be used to designate them.[1]

Cementum is specialized bone; it replaces the bone of attachment of the more primitive forms. Two kinds are described,[2] the one, acellular; and the other, cellular, which more nearly resembles the parent bone.

The acellular type covers practically all of the root and is a thin hyaline layered structure. It easily can be entirely removed when the teeth are scaled. Both fiber bundles and individual fibers are embedded within its substance. It is a slowly and continuously growing structure, new layers being apposed from within outward. A cementoid layer is usually visible just under a layer of cementoblasts.[3, 4]

The cellular type varies greatly in thickness and is observed most frequently in the apical areas of roots and in their bifurcations. It, too, is a layered structure, containing irregularly arranged lacunae with cementum corpuscles. Only the more superficial corpuscles are viable; the deeper ones degenerate when they become more remote from nutritive sources. This suggests a low order of metabolic activity for cementum and implies that only the surface lamellae are vital.[3, 4] Under abnormal stresses a spike-like hypercementosis develops to effect a better hold on fiber bundles, in order to prevent their being torn away from their attachments.[3-6] This compares with a similar reaction in bone; for example, the

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thickening of bony ridges under musculotendon attachments in extreme physical employment.[7]

Apparently an attachment apparatus of a certain size is essential for tooth function.[2-6, 8-17] When alveolar bone is lost in disease, or the bone is weakened as in Paget's disease, or the root is shortened as in root resection, in root fractures, in loss of periodontal attachment as in granulomas, and in the orthodontic movement of teeth, new layers of cementum are formed on the teeth, from several lamellae to extreme bulbar types of hypercementosis, to make up for the amount of lost attachment and to keep the tooth functioning. This accounts for most instances of hypercementosis. There are instances of hypercementosis, however, that cannot be explained.

THE PERIODONTAL MEMBRANE

The periodontal membrane occupies the space between the roots and alveolar bone. It is a highly vascular, dense, collagen fibrous membrane. The fibers are mostly arranged in bundles of irregular shapes and sizes. These are the principal fibers. They are embedded in bone on the one side and in cementum on the other. Between the bundles are the loosely arranged indefinite collagen fibers, which support the vascular tree, nerves and lymphatics.

G. V. Black[18] first recognized the principal fiber bundles and classified them: (1) free gingival fibers, (2) transseptal fibers, (3) alveolar crest fibers, (4) horizontal fibers, (5) oblique fibers and (6) apical fibers.

The free gingival groups extend from cementum into the gingivae. They are not attached tooth-to-bone; hence these groups are not a part of the true attachment apparatus.

The transseptal groups are attached tooth-to-tooth, from the mesial surface of one root to the distal surface of an approximating tooth. This group is remarkably persistent.

The alveolar crest fibers are a small group extending in a fan-like fashion from the bony crest crownward to cementum, chiefly on the labial, buccal and lingual surfaces. The horizontal group is small, located just below the alveolar crest and transseptal fibers, and is radially arranged. The oblique fibers constitute the largest group and are attached higher on their bony attachment than on their cemental attachment, at about a 45 degree angle. Stresses on teeth therefore impart pull to bone. This is important, for pull (tension) stimulates new bone formation. The apical fibers fan out from horizontal on the lateral aspects of a root tip to vertical in the fundus of the alveolar socket. Altogether these fiber bundles are designed in their varying arrangements to resist to a degree the vertical and horizontal forces of mastication.

The principal fibers have microscopic undulations along their course which become taut under stresses, permitting slight individual tooth movements. Between and around the fibers are fibro-

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18. Black, G. V. A study of the histologic characters of the periosteum and periodontal membrane. Chicago, W. T. Keener Co., 1887.

blasts, whose shapes are modified because of pressure. In the indefinite fiber areas are found blood vessels, lymphatics[19] and nerves.[11,12,20] The blood vessels arise from three sources:[2,5,21] from the periosteum of the cortical bone to and through the gingivae, from the bone marrow through lateral perforations of the alveolar bone, and from periapical vessels. They form an elaborate anastamosing network. They may also anastamose with the pulpal vessels through accessory foramens or at the apical area, which might explain pulpal involvement as a complication in periodontal disease. These vessels are supplied with their own sympathetic nerve system. Many of these vessels are large and a hydraulic type of resistance[14] to stress has been ascribed to them. The lymphatics form a complicated pattern of capillaries that drain into the various cervical lymph nodes, which explains lymph node enlargement in dental infections. The nerves are both myelinated and naked.[19-29] Their endings have been described as knob-like swellings, rings or loops around fiber bundles, and free endings between fibers. They are proprioceptive and the sense of localization is imparted through them. All the cells of defense and of repair are also found here when occasion demands.[13]

On the bony side are osteoblasts and on the cemental side are cementoblasts. In repair and adjustments, osteoclasts also are found. In addition are found epithelial rests and cementicles. The epithelial rests are remnants of the sheath of Hertwig,[10] which in early tooth formation are found in long strands; they are eventually broken up into smaller rests. However, in many persons they persist in net-like arrangements. After trauma and infection they can proliferate and change from simple epithelium to cysts and tumors.

Cementicles[2] are calcified bodies of varying shapes and can be found in almost any area of the periodontal membrane, especially near enamel droplets. Some remain free; others become attached to cementum and alveolar bone. They are said to be dystrophic calcifications.

The periodontal membrane has four functions: formative, in that it can tear down and rebuild new cementum and bone; supportive, in that it maintains the tooth when resisting stresses; nutritive, in that it furnishes all necessary substances for the maintenance of metabolic activities; and sensory, in that it receives impulses and reacts to stimuli.

ALVEOLAR BONE

Alveolar bone is the supporting bone of the teeth. It is a transitory structure, for it does not develop in anodontia and mostly disappears after the teeth are lost. In a fully erupted and functioning tooth, the bone to which the periodontal fibers are attached is found to be a cribriform plate. Depending on the function of the teeth, these cribriform plates are supported by bony trabeculae, well formed and numerous in functioning areas and thin and scanty in nonfunctioning areas. One or more large arteries, veins and nerve bundles are longitudinally situated in the

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interradicular bony process, and branches from them enter the periodontal membrane through the many openings in the cribriform plates.

All the bones of the body change throughout life.[7] Probably alveolar bone is required to change more often than other bones. The development and eruption of teeth, the shedding of deciduous teeth, the loss of permanent teeth, wear and tear, all demand alterations and adjustments in alveolar bone. It requires considerable time and space for the construction of the adult pattern of bone. During periods of transition, a temporary bone (bundle bone) is formed.[29] Later, this is reconstructed and becomes more permanent lamellated bone. However, in the almost unlimited episodes of bone reconstruction, lamellated bone itself is occasionally removed to make way for another construction.

Alveolar bone is as subject to Wolff's law as are other bones. In a well-functioning tooth the alveolar bone and supporting trabeculae are thick, whereas in a nonfunctioning tooth a disuse atrophy occurs. Here the cribriform plate and the supporting trabeculae are frail and scanty.

A similar law may be postulated for the periodontal membrane.[6, 22-25, 27, 30] In good function this membrane is dense and wide; when not functioning it is thin and atrophic. New cementum on the root or new bone on the cribriform plate or both may contribute to make the periodontal membrane narrower.

The attachment apparatus is designed to permit individual tooth movements. When a bony socket is examined and critical measurements are made of the thickness of the periodontal membrane, it is noted that the socket has an hourglass appearance.[9, 23, 25] The periodontal membrane is widest at the bony crest and narrowest just below the center of the root; it widens again in the apical region. This indicates a tipping motion, which can be accentuated in experimental work and in orthodontic tooth movement, resulting in resorption on the pressure side and deposition of bone and cementum on the tension side.

After many years of use, contact points of teeth are worn to contact planes. Nature has designed a compensatory mechanism to tighten up the resulting slack; for example, the physiologic drift of teeth. This follows a medial direction.[28, 29] Examination of a tooth socket almost always reveals resorption on the mesial aspect, with new bone being built on the distal aspect. Resorption of the mesial aspect (pressure) is by osteoclasts, rebuilding of new bone on the distal aspect (tension) is by osteoblasts. This drift is not continuous, but in waves governed by the amount and rapidity of contact point abrasion. Sometimes new cementum forms on the distal aspect, so that a distal root surface shows a thickening of the cementum. Consequently, the distal periodontal membrane space does not move so far as a mesial periodontal membrane space.[15] The new bone on the distal side of the socket is at first bundle bone; when this attains a certain thickness, reconstruction occurs, with replacement by lamellated bone.

Often in this physiologic drift epithelial rests remain behind. These are later found in the marrow spaces and can be used as an index of the amount of drift that has occurred. While this drifting occurs, many blood vessels are also left behind; most of them are incorporated in the oncoming bone. However, on the mesial aspect, the tooth finally meets with some of the larger vessels. These remain in the periodontal membrane and may be a source of severe hemorrhage. Occasionally resorption of cementum occurs as the tooth encroaches on these vessels.

As the teeth are worn by attrition, they erupt further into the occlusal

30. Kronfeld, R. Histopathology of the teeth and their surrounding structures. Philadelphia, Lea & Febiger, 1933.

plane.[1, 2, 5, 7, 8, 18-30] In order to maintain a constant root length, new cemental layers are formed on the root tip. When such an eruptive movement is accelererated, new bone fills the fundus of the socket, resulting in a shortening of the attachment apparatus. Sometimes both new cementum on the root tip and new bone in the fundus of the socket are found.

Teeth are subject to many injuries, particularly trauma. Either periodontal fibers are torn away or tears in the cementum occur.[8] When only fibers are torn, the cementum and bone holding them are resorbed; then new fibers are aligned and new cementum and bone are laid down in the resorbed areas to reconstruct a new attachment.[28] Most often the new cementum is cellular; occasionally it is acellular; sometimes it is both. Such reattachment cannot take place if the epithelial attachment proliferates to cover the cementum.

When injuries of cementum occur, a wide variety of patterns of adaptability follows.[8] After the occurrence of cemental tears, root fractures, or resorptions on the lateral surfaces of the roots, a modified attachment apparatus is created in response to functional demands. This implies that teeth that are being treated should be subjected immediately to functional stresses. In rare instances ankylosis[16, 17] may take place, especially in deciduous teeth that have been retained too long.

SUMMARY

An attempt is made to show that the attachment apparatus is a capable structure than can compensate to the same degree, and better than most other body structures, in response to injuries. A good understanding of these potentialities is of considerable aid in the treatment of periodontal diseases.

The epithelial network in the periodontal membrane

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The epithelial remnants in the periodontal membrane are rather neglected structures. It is known that these epithelial islands develop from the enamel organ during root formation and eruption from the so-called Hertwig's epithelial root sheath. It has been shown that the epithelium forms a continuous network in the periodontal membrane. Only in a microscopic section do these structures appear in the form of islands, rests or remnants, as they are called, but these are only cross sections of epithelial cords forming the network. In a complete reconstruction or tangential section (Fig. 1), the true structural arrangement becomes evident.

The function of the epithelial root sheath is known. It has been ascertained that the epithelium exerts an organizing effect on the mesenchymal dental papilla (pulp), inducing the development of odontoblasts and, thus, the dentin. Without the presence of the epithelial root

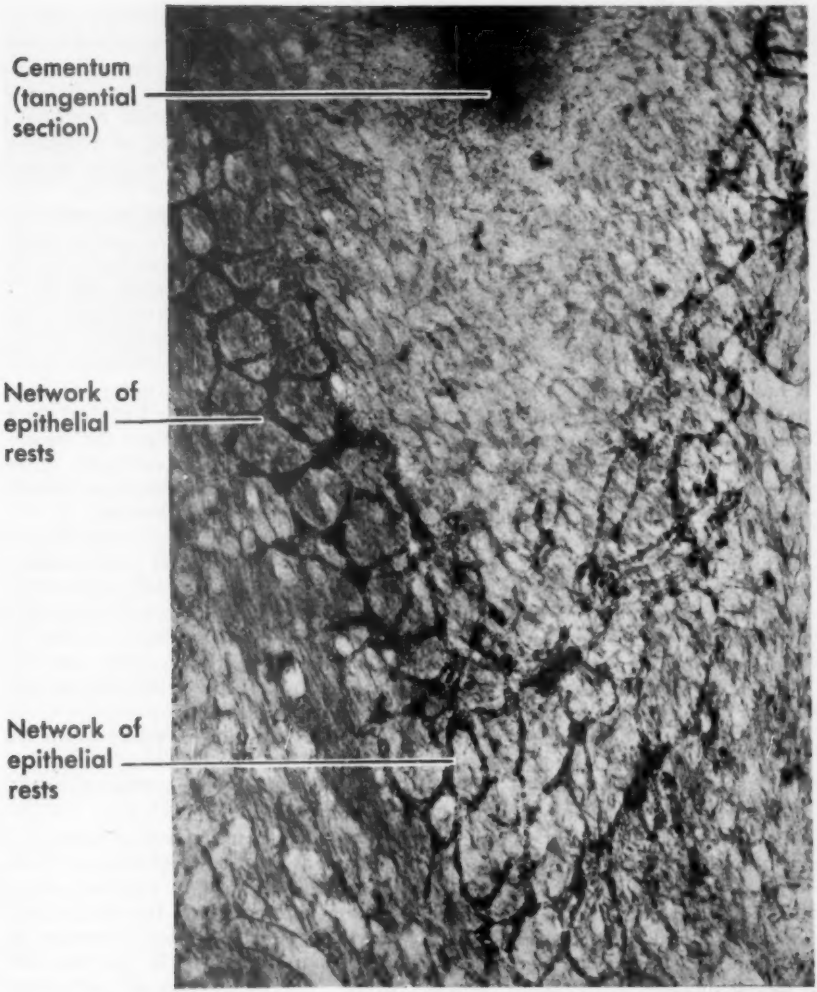

Fig. 1 • Network of epithelial rests in periodontal membrane

sheath and its organizing effect, no dentin, and hence no root formation, could occur.

The function of the epithelial network in the periodontal membrane, however, is unknown. No definite evidence has been produced to prove that this structure has any function. It is hardly conceivable that epithelial tissue, having no function to perform, would remain vital throughout the life span of an individual. These epithelial cells must undergo degenera-

tion, as they reach the end of their life span (20 to 30 days or slightly longer). To be found at all ages, these cells must regenerate by mitotic activity. However, such regeneration can be expected only if required by function.

The formation of cementum on the surface of the developing root cannot take place until the epithelial root sheath is broken up and connective tissue proliferates between the surface of the dentin and epithelium.

By analogy, it could be assumed that the epithelium in the periodontal membrane has a similar biologic function as it has at the forming root end. At the root end, the epithelium stimulates the cells of the pulp to produce odontoblasts; in the periodontal membrane, it might have the function of stimulating the formation of cementoblasts, and thus, cementum.

This idea is not new; it has been suggested before, but has never been proved, and unfortunately, no one seems to pay any attention to this important problem. The continuous formation of cementum, which is the most important biologic feature of the cementum (Gottlieb[1]), may depend not only on the presence, but also on the functional ability of these epithelial structures. The biologic difference between the cementum (continuous apposition) and the bone (alternating resorption and formation) may be due to the function of this epithelial network.

There are a few observations available which would indicate that the epithelium in the periodontal membrane does have a special function. For example, herbivorous animals, whose teeth are vital organs, have an extensive cementum formation and a rich network of epithelium in the periodontal membrane. In carnivorous animals, where the cementum is rather thin, the epithelium is sparse.

It has often been observed that teeth, once extracted and replanted, become ankylosed. This means that the tooth, as such, has lost its organ character and the surrounding tissue reacts to the replanted tooth just as it would to a bone implant. This biologic reaction may be caused by the absence of the epithelial network in the periodontal membrane, which was lost in the act of extraction. If only a small portion of the periodontal membrane is lost, as in necrosis from trauma, repair is possible, probably because of the influence of nearby intact regions. However, ankylosis has been observed in traumatized regions.

The number and the condition of epithelial remnants in the periodontal membrane present a problem which has never been resolved. In specimens (human autopsy material) which I studied there were indications that where the cementum was thick or hypertrophic, there were more epithelial remnants present than in cases with thin cementum.

An interesting finding in this respect is that in an early case of periodontosis,[2] large epithelial islands were observed in the degenerated periodontal connective tissue. These epithelial islands seemed to be hypertrophic. It is possible that the hypertrophy of the epithelium in this case was a compensatory overgrowth, the epithelium exerting a futile effort to stimulate the degenerated connective tissue to produce cementoblasts and thus, cementum.

One could go a step further in speculation and assume that degeneration of the epithelium can lead to cessation of cementum formation and thus lead to the development of pathologic processes in the supporting tissues. It has been observed that periodontosis sometimes affects a group of teeth such as the first molars and incisors. It has also been observed[3] that sometimes these teeth, af-

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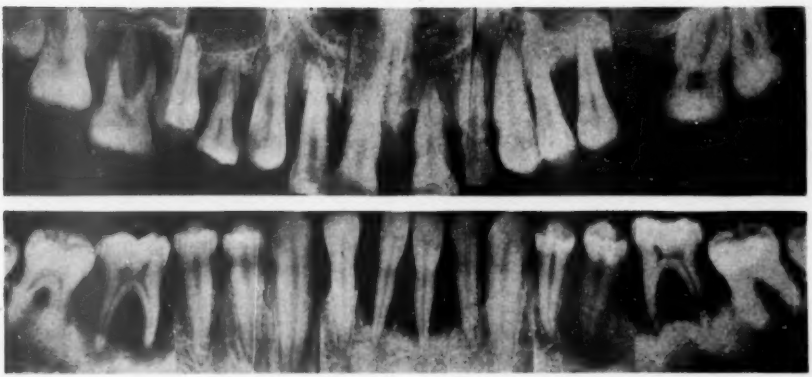

Fig. 2 • Showing extensive bone resorption in incisor and first molar regions

fected by periodontosis, show signs of enamel hypoplasia. Enamel hypoplasia is a disturbance in the formation of the enamel and follows a chronologic developmental pattern; that is, it affects all regions which develop at the time of the disturbance. It can be assumed that the primary factor involved has disturbed the enamel epithelium. It is possible that this same factor, or some other, as well, has led to a disturbance in the formation and function of the epithelial root sheath. Often, in these cases, the roots of the affected teeth appear rather thin (Fig. 2). If the periodontal tissues are unable to cope with functional tissue changes, a breakdown can occur at an early age. The continuous formation of cementum is a necessary factor in this functional adaptation. The pathogenesis of this type of periodontosis might have its basis in a developmental disturbance.

It is to be hoped that interest will be stimulated in further studies, especially those of an experimental nature, to discover the function of the periodontal epithelial network.

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Acquire Mental Knowledge • Do not imagine that the knowledge, which I so much recommend to you is confined to books, pleasing, useful as that knowledge is. But I comprehend in it the great knowledge of the world, still more necessary than that of books. In truth, they assist one another reciprocally; and no man will have either perfectly who has not both. The knowledge of the world is only to be acquired in the world, and not in a closet. Books alone will never teach it to you; but they will suggest many things to your observations which might otherwise escape you; and your own observations upon mankind, when compared with those which you will find in books, will help you to fix the true point. *Chesterfield, "Letters to His Son."*

Pathology of periodontal diseases

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Periodontal diseases are responses to injuries either separate or in combination, to the gingiva, periodontal membrane and alveolar bone. While local conditions have some effect on the tissue changes which result from injury, the underlying pathologic processes are similar to those that occur in other tissues of the body. These basic pathologic reactions are inflammation, degeneration, atrophy, hypertrophy and hyperplasia. In addition, there may be pathologic alterations in the osteoblastic and osteoclastic activities. It is important that these pathologic processes be understood if diseases of the periodontium are to be given intelligent consideration.

INFLAMMATION

Inflammation is a response of tissue to injury. It is part of the body's protective mechanism and tends to hold in check, eliminate or destroy injurious agents. It usually is followed by repair, which some authorities consider inseparable from inflammation. If inflammation and other protective responses of the body are successful, disease is eliminated but if these body defenses are unsuccessful the tissue is destroyed and death occurs either locally or throughout the entire body. In general, therapy is directed at aiding the inflammatory process by such means as reducing the ability of the bacteria to multiply, by removing irritants from tissues, or by neutralizing toxic materials. When inflammation is regarded in this manner it is understood as a beneficial and protective mechanism. Unfortunately, because inflammation occurs as a reaction to injury, the inflammatory process all too often is considered as an injurious process rather than as the beneficial process that it actually is.

Inflammation not only is a response to invasion by living organisms such as bacteria but, likewise, may be activated by physical trauma, chemical agents or radiant energy. Bacteria and inanimate types of destructive influences always are present in the oral cavity. Overhanging margins of restorations, improperly used toothbrushes and toothpicks, and dental calculus are a few of the physical agents which must be considered. The acids, alkalies, heavy metals and many of the irritating foods, as well as tobacco and alcohol, are chemical agents which adversely influence the oral tissues. Injurious radiant energy includes heat which comes from smoking, hot foods and liquids, roentgen rays and atomic energy. Under the influence of any of these injurious agents, the body may set up inflammation as a defense.

It is necessary to understand the dynamic process of inflammation if periodontal diseases are to be understood. If an area were observed immediately after it had been injured, a momentary vascular contraction would be seen, followed almost immediately by dilation of the

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blood vessels and the appearance of many tiny small blood channels where collapsed endothelial tubes had existed previously. Shortly thereafter, there is an increase in the permeability of the vessel walls, an increase in the intercapillary blood pressure and a change in the osmotic pressure. As a result, an exudate of fluid and cells passes through the vessel walls and enters the injured tissues surrounding the vessels. This is not a simple filtrate of blood but the fluid contains many large molecules which ordinarily do not escape from the vessels. As a result of the exudation of cell-free fluid from the vessels, there is an increase in the viscosity of the intravascular blood locally, with slowing of the circulation. The capillary filtrate which escapes from the vessels accumulates in the tissue spaces, producing edema. The fluid which escapes from the capillaries does not return to the blood vessels but is drained away by the lymphatics with a resultant increase in the flow of lymph. The cells within the blood streams adhere to the vessel wall and soon begin to escape from the vessel. The movement of the leukocytes through the vessel walls is by ameboid activity. In each cubic millimeter of blood there are approximately 4,000 cells, each of which is capable of independent ameboid movement. The ameboid cells are attracted to the area of injury by chemotactic substances which include the bacteria themselves and the products of injured tissues. Among the best recognized of these leukocyte-attracting substances is leukotaxine which is a polypeptide that has been isolated from areas of injury.[1] In addition, other enzymes such as hyaluronidase may prepare the tissues for penetration by the fluids and cells. The cells which appear in the areas of inflammation not only come from the blood stream but also from the reticuloendothelial system (chiefly lymph nodes) and from the tissues in the region of inflammation.

In early states of inflammation, the predominant cell is the polymorphonuclear neutrophilic leukocyte or microphage which is capable of motility (ameboid movement). A second phagocytic cell found in the tissues is the macrophage, which comes from the blood stream as a large monocyte, or from the tissues as a resting-wandering cell. The reason that these cells are termed "phagocytes" is because they are capable of ingestion of particulate matter. This process of phagocytosis probably occurs as a result of the difference in interfacial tensions between the phagocytes, the particles and the surrounding fluids. Phagocytosis is extremely important in removal of foreign particles, obnoxious formed particles, and bacteria. In an area of inflammation, lymphocytes and plasma cells also appear and play important parts in the defense reaction. These cells usually are not found in the earliest stages of inflammation but become more prominent in later phases.

The lymphocyte, like the polymorphonuclear leukocyte, is derived at first from the blood stream, but it may also migrate from lymphoid tissue present in the region. The function of the lymphocyte is not known exactly but it has been suggested that it may act as a storehouse for nucleic acids which act to neutralize toxic materials produced in the breakdown of the proteins of the tissues.

The plasma cells are characteristic of chronically inflamed tissues, but are seen in most inflammations in the oral region. The exact origin of the plasma cell has not been determined definitely but is considered as derived from the lymphocyte, the monocyte, or from primitive connective tissue cells. The most important function of the plasma cell probably is to supply the material for antibodies which neutralize the injurious antigens.[2]

In addition to the neutrophilic poly-

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morphonuclear leukocytes, macrophages, lymphocytes and plasma cells, a few eosinophilic and basophilic polymorphonuclear leukocytes are found in the regions of inflammation. The eosinophils are found more prominently in states of hypersensitivity and in regions in which animal parasites are present. It has been suggested that the chief function of the eosinophils is to produce an antihistamin-like substance but this viewpoint is not held in high regard at the present time. The basophils may be the sources of heparin which prevents the formation of massive clots within inflamed areas.

Inflammation, then, must be considered as a complicated process which is carried out by fluids and by numerous cell types. The fluid, and each of the cell types, have specific assignments. Clinically, the phenomena of inflammation are characterized by redness (hyperemia) and warmth, which are due to the increased blood supply in the part; swelling (edema), which is the result of the increased fluid content of the tissues; pain, which results from the irritation of nerve endings by toxins or by physical pressure; and interference with function. Inflammatory responses vary with the injuring agents and the tissue involved. Among the varying responses are fibrinous, purulent, serous and catarrhal inflammations. In some inflammatory lesions proliferation is characteristic, while in others necrosis occurs. Inflammations of the periodontal tissues, including the gingiva, may be catarrhal, fibrinous, purulent or serous, and they may produce proliferative changes. When the surface continuity is lost and there is inflammation beneath, the lesion is an ulcer.

REGRESSIVE CHANGES

Injured tissues commonly undergo regressive changes which include degeneration, atrophy and necrosis. These changes may be produced by various agents, including microorganisms, trauma, chemical agents, radiant energy and, in addition, metabolic disturbances. The retrograde changes are often, but not necessarily, accompanied by inflammation.

ATROPHY

Atrophy is a decrease in the size of organs or cells which once were normal in size. Atrophic organs and cells are differentiated from those which are hypoplastic (not fully developed) or aplastic (almost completely undeveloped), and from agenesis (completely absent) due to developmental failure. Atrophy may be due to reduction in the size or number of cells, or both. Basically, atrophy is caused by inadequate nutrition of the cells. Among the recognized causes of such nutritional failure are disuse, pressure, inadequate blood supply and starvation. Physiologic atrophy is a type of regression which occurs in the thymus at puberty, in the ovaries and uterus at the menopause, and in many tissues as a result of the aging process. While atrophy usually results in reduction in size of the organ, it is possible the atrophy cells may be replaced by connective tissue or fat. Atrophic changes in the periodontium reduce the support of the teeth, especially in elderly persons.

DEGENERATION

Degeneration is a term applied to reaction of cells to injury with swelling and accumulation of undesirable substances within their cytoplasm. Degenerations result from infections, intoxications, metabolic disturbances and other causes. Most degenerative changes are reversible but certain severe degenerative processes lead to cell death (necrosis). The type of degeneration varies with the type of tissue and the cause of the change. Among the retrograde changes are parenchymatous (cloudy swelling), hydropic, fatty, hyalin, mucinous, amyloid and collagenous degenerations.

NECROSIS

Necrosis is cell or tissue death within the living body and usually is a sequel of degeneration or of inflammation. A severe degenerative process preceding necrosis is called necrobiosis. There are various types of necrosis, including coagulation, liquefaction, caseous, gummatous and fat necrosis.

GANGRENE

Gangrene is massive necrosis usually accompanied by bacteria invasion. Dry, moist, and gas gangrene are among the recognized types.

HYPERTROPHY AND HYPERPLASIA

An increase in the size of tissue can be produced either by hypertrophy (increase in the size of each cell) or hyperplasia (increase in the number of cells) or by both phenomena together. Ordinarily, normal growth occurs through the same processes but the terms hypertrophy and hyperplasia usually are used to infer abnormal enlargement of a part such as hypertrophy and hyperplasia of the gingiva. Whether an enlargement of the tissue results from hypertrophy or hyperplasia is determined by microscopic examination of its cells. It is usually extremely difficult to determine clinically whether a tissue is enlarged from hypertrophy or hyperplasia or both. For this reason the terms are often used interchangeably.

BONE ACTIVITY

Bone is extremely plastic tissue and is undergoing deposition and resorption throughout life. When deposition and resorption are equal the bone is maintained at status quo. When bone is developing or undergoing enlargement, deposition (osteoblastic activity) exceeds resorption (osteoclasia). Bone may be reduced by maintaining normally osteoblastic activity and increasing osteoclasia or by reducing osteoblastic activity and maintaining ordinary osteoclastic activity.

GINGIVITIS

Gingivitis is the general term applied to inflammation of the gingiva. Gingivitis usually is the result of injury by locally active agents. Calculus at the gingival margin and apical to the gingival margin, malaligned teeth with poor contacts, faulty restorations with rough or overhanging margins, poor contours, inadequate contacts or impinging clasps, and trauma from improperly used toothbrushes, toothpicks and dental floss are among the more important physical agents. Strong chemicals in beverages, foods, tobaccos and drugs also may be etiologic factors. When any of these agents injure the gingiva, invasion by living agents follows. The oral fluids surrounding the gingiva, and especially within the gingival sulci, contain many pathogenic and potentially pathogenic bacteria. Streptococci, staphylococci, diplococci, fusiform organisms, spirochetal forms, vibrios, diphtheroids, actinomycetes, leptothrix, amebae, yeasts and herpetic virus may all be isolated from apparently normal mouths. Whenever the continuity of the epithelial covering is lost these organisms find it easy to invade the underlying connective tissue. The first line of defense against injury of the gingiva, then, is its covering epithelium. The gingival epithelium may be nonkeratinized, keratinized, or parakeratinized. Brushing appears to increase the keratinization of the gingival epithelium which is exposed to the toothbrush and thereby increases resistance to invasion. However, the epithelium lining the gingival sulcus or a gingival pocket shows neither keratinization nor parakeratinization. Once the protection of the oral mucosa is broken only the second line of defense is left. Primarily, this includes the inflammatory proc-

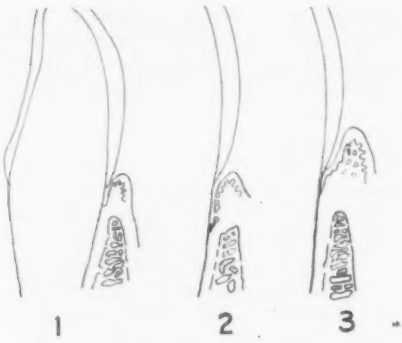

Fig. 1 • Production of pockets of periodontal type and pericoronal type. 1. Epithelial attachment is on enamel and cementum and alveolar bone crest is normal. 2. Periodontal pocket with ulceration of pocket (sulcus) epithelium, proliferation of epithelium apically on cementum and loss of alveolar bone crest. 3. Pericoronal pocket resulting from enlargement of gingiva (edema, hyperemia, fibrosis, or neoplastic infiltration) with epithelial attachment and alveolar bone crest normal

ess, the antigen-antibody reaction and repair.

The most important single defensive barrier against the great number of living agents in the gingival sulcus is the sulcus epithelium. It is in a position where it must withstand irritation that is produced by abrasion from calculus, by food impaction, and from overhanging margins of fillings. In the final analysis, calculus is the most important single factor in gingivitis, but it must be remembered that the resultant injury from the calculus only opens the way for invasion by bacteria of the oral cavity.

The clinical type of gingivitis varies depending on the number of interacting factors. The type of injurious agents may somewhat modify the resulting inflammation, as may also certain local tissue influences and systemic factors. The gingiva may be redder than normal (hyperemic), or pale. It may be firmly distended or swollen and soft (edematous). The swelling may be generalized or may be confined to the interdental papillae and the gingival margins, and the gingival sulcus may or may not be increased in depth. The only way in which gingivitis, *per se*, increases the depth of the gingival pocket is by enlargement of the gingival margins through edema, cellular infiltration, or stimulation of cell growth (Fig. 1). The gingival sulcus may be deeper than normal in some individuals with gingivitis without enlargement of the gingival margin, but this is the result of pre-existing or coexisting periodontitis.

Calculus (Fig. 2), overhanging margins and oral filth usually result in ulceration of the epithelium lining of the sulcus followed by thickening of the gingival margins, enlargement of the dental papillae, loss of density and increase in the depth of the gingival sulcus, all caused by edema. The normal pale pink of the gingiva is increased to a deeper pink or red as the result of hyperemia. Examination of such a tissue would show enlargement of blood vessels, infiltration of the cell and intracellular spaces by fluid and the presence of large numbers of polymorphonuclear leukocytes, a few plasma cells and occasional lymphocytes, and possibly some macrophages.

The defensive mechanism is active in these cases and if the injurious agent is removed, it is highly probable that the tissue will repair and return to normal. If it is neglected, however, the periodontal membrane and alveolar bone will be involved.

There are varying types of gingivitis, clinically, as a result of different injuries and tissue reactions. Among these are necrotizing ulcerative gingivitis which is characterized, clinically, by the presence of ulcers, necrosis, and pseudomembrane (Fig. 3). Histologically, the most striking characteristic would be the presence of fibrinous exudate over the ulcers. Another type of gingivitis which may be a sequel to marginal gingivitis, or may be primary, results from irritation with stimulation of cell production. It is gen-

erally classified as hypertrophic or hyperplastic gingivitis. Hyperplastic and hypertrophic gingivitis must be differentiated from hypertrophy and hyperplasia of the gingiva which result from hereditary factors (Fig. 4), diphenylhydantoin sodium (Fig. 5), hormonal imbalance (Fig. 6, left) or leukemias (Fig. 6, right). When there is a superimposed disbalance of estrogenic hormones the type of gingival change which is seen is described as desquamative. The surface epithelium is somewhat atrophic and the basement membrane is reduced, or absent. As a result the attachment of the epithelium to the underlying connective tissue, in which the ground substance is disturbed, is extremely poor and the epithelium readily desquamates, or an air-filled bleb may be formed by applying air under pressure.

It should be remembered that gingivitis is an inflammation of connective tissue and epithelium, usually a sequel of ulceration of the epithelium followed by bacterial invasion.

PERIODONTITIS

Periodontitis is an inflammatory disease of the periodontium. Not only is the gingiva injured but the submucosa around the alveolar bone crest and the cementum are secondarily involved in this process. As the sulcus epithelium is ulcerated the epithelial attachment usually is stimulated and proliferates apically, resulting in the deeper sulcus now called a periodontal pocket (Fig. 7). The depth of the pocket is not equal around the tooth so that the pocket may be fairly narrow.

Early in the process the pressure of the edematous tissue around the alveolar bone crest and enzyme activity result in resorption of the alveolar bone crest. With this the alveolar crest fibers are lost and at about the same time the free gingival and transseptal fibers are destroyed. As a result, the gingival tone is lost, favoring food impaction and further injury to the sulcus epithelium. The inflammatory process proceeds along the course of the blood vessels into the bone marrow spaces and continues to invade the periodontal membrane at its most coronally disposed aspect. Soon the horizontal and oblique fibers are reduced and the stress on the remaining fibers and their alveolar bone are increased. The inflammatory process and accompanying derangement of bone stresses result in reduced activity of the periodontal membrane in its role as periosteum and pericementum.

Actually, the greatest and most severe effect is that on the periodontal membrane which is the supporting apparatus

Fig. 2 • Gingivitis as seen microscopically. Calculus (Ca) has caused ulceration of sulcus epithelium followed by bacterial invasion and inflammation. The epithelial attachment (EA) is still present. Early resorption (R) of alveolar bone crest indicates progression into periodontitis. (From photographic collection of Rudolf Kronfeld)

Fig. 3 • Necrotizing ulcerative gingivitis showing fibrous inflammation with ulceration and necrosis of gingival papillae and margins

Fig. 4 • Pericoronal pockets associated with hereditary fibromatosis. Poor oral hygiene is a complicating factor

Fig. 5 • Gingival enlargement caused by diphenylhydantoin sodium therapy. Enlargement is confined to the gingiva. After this fibrous enlargement, inflammation may occur in the pericoronal pockets or in clefts such as that seen at the lower right lateral incisor

of the tooth as well as a periosteum and pericementum. Continuous deposition of cementum ceases, osteoclastic activity overshadows the osteoblastic activity on the alveolar bone side and the principal fiber bundles are disrupted. The vitality of the cementum, often mentioned as an important factor, is of little consequence as the cementum is simply a product of the periodontal membrane and may be deposited as long as the periodontal membrane is capable of function and ample stimulus is afforded. Bone reduction in periodontitis usually is the result of maintaining a normal, or slightly reduced, osteoblastic rate, while the osteoclastic activity is increased.

PERIODONTOSIS

Periodontosis is a degenerative disease of the periodontium. This relatively rare

disturbance usually follows systemic disease although doubt has been cast on this theory.[3, 4] The primary effect on the periodontium is reduction in all the metabolic (generative) processes. Inflammation is not a factor. With reduction in activity of the connective tissue of the periodontium the continuous production of cementum is greatly minimized, or ceases entirely, while osteoblastic activity on the other border of the periodontal membrane is practically halted. As a result the attachment of the Sharpey fibers is lost, the principal fiber bundles degenerate, bone stimulus ceases, the alveolar bone is resorbed, and the periodontal membrane widens (Fig. 8). Roentgenographically, the lamina dura disappears and the bony support of the teeth is reduced (Fig. 9) and clinically, the teeth are lost and tend to space (Fig. 10).

ATROPHY

As a result of abnormal stress, disease, trauma, aging, or unknown factors (precocious senile atrophy) the periodontium may atrophy. Atrophy may follow the inflammatory process. Although the reduction of the periodontal tissues may be a result of aging, and as such be a physiologic process, it is more probable that such atrophic changes are the result of repeated stress and trauma.

HYPERTROPHY AND HYPERPLASIA

Hypertrophy and hyperplasia may enlarge the gingiva (Fig. 1, 2, 3) and cause pericoronal pockets. Among the causes are inflammation (Fig. 11), hereditary fibromatosis (Fig. 4), hormonal imbalance (Fig. 6, left), leukemia (Fig. 6, right), and use of diphenylhydantoin sodium to treat epilepsy (Fig. 5). The reaction varies. In inflammation the enlargement usually is not true hypertrophy or hyperplasia but edema and hyperemia; in hereditary fibromatosis, and diphenylhydantoin sodium hypertrophy, there is a great increase in the connective tissue;

3. Ramfjord, S. Effects of acute febrile disease in the periodontium of rhesus monkeys with reference to poliomyelitis. J. D. Res. 30:615 (Oct.) 1951.

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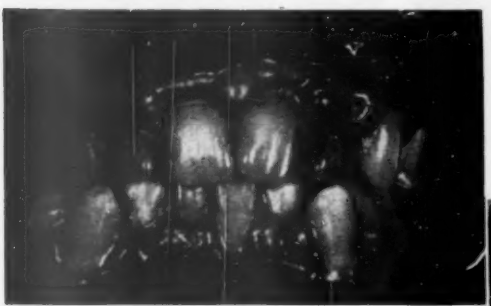
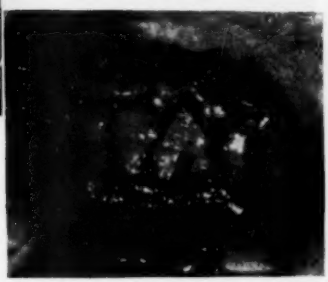


Fig. 6 • Left: Gingival enlargement caused by hormonal imbalance in 21 year old woman. Traumatism is complicating the clearer entity of vascular enlargement



Right: Gingival enlargement associated with leukemia

in hormonal hypertrophy the reaction is chiefly vascular; while in leukemia the tissues may actually be engorged by leukemic cells transported by the blood stream.

TRAUMATISM

Traumatism is not a pathologic process but is a mechanical process causing certain tissue changes in the periodontium. Trauma from improperly used toothbrushes, toothpicks, dental floss, dental tape, sharp instruments, pencils, pins, bones, and other agents may ulcerate the epithelium and open the way for infection. However, traumatism in connection with periodontal disease usually is thought of as the result of occlusal disequilibrium.

Under abnormal occlusal stresses the periodontium may be injured, osteoclastic activity may overshadow osteoblastic activity on the side of pressure and undesirable changes occur in the tissues.

INTERRELATIONSHIP OF PATHOLOGIC PROCESSES

It is uncommon for diseases of the periodontium to exist independently of each other. A single periodontium may exhibit evidence of periodontitis with periodontosis, atrophy, hypertrophy and hyperplasia, or traumatism; or any combination of these may exist. Unfortu-

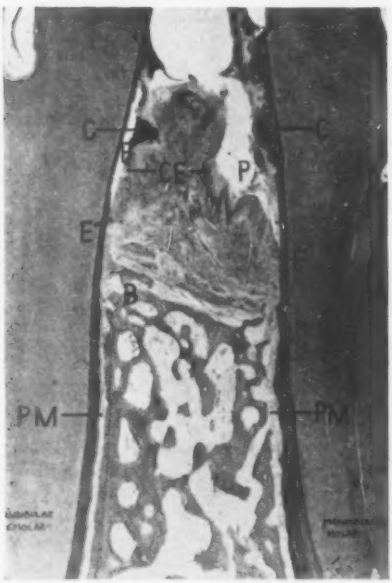


Fig. 7 • Pathologic picture in periodontitis. Calculus (c) has resulted in injury to the crevicular epithelium (CE). Inflammation which followed has involved the alveolar bone crest and the coronal portion of the periodontal membrane (PM). (Courtesy of Balint Orban)

Fig. 8 • Pathologic picture in periodontosis. The periodontal membrane has degenerated with resultant loss of alveolar bone (lamina dura) on the right. The principal fiber bundles have become indefinite. Calculus in the gingival sulcus is a complicating factor as possibly is trauma. (Courtesy of Balint Orban)

Fig. 9 • Maxillary incisors in patient with periodontosis associated with uncontrolled diabetes. Compare with Figures 8 and 10

nately, the coexistence and interrelationship of these periodontal diseases have been overlooked. A single diagnosis may lead to improper treatment. All factors causing pathologic changes should be identified and these factors are often traced through proper interpretation of symptoms and their relationship to pathologic processes.

REPAIR

The possibility of repair in the periodontal tissues is relatively good. While the results of treatment in any clinical case will depend on many factors, some under the control of the dentist, and others, consciously or unconsciously, the responsibility of the patient.

The question of reattachment of periodontal tissues has been debated for many years.[5] There can be no doubt, pathologically or histologically, that the periodontal fibers can regenerate after periodontitis has been treated successfully and all adverse effects either removed or reduced to inconsequence. These same fibers are destroyed and reformed in orthodontic treatment and in children whose teeth have been driven back into the sockets by trauma. Following surgical procedures their regeneration is taken for granted. Linghorne and McConnell[6] have demonstrated repair of periodontal structures histologically. The expected sequence of events is illustrated in Figure 12.

5. Leonard, H. J., and others. Reattachment: a symposium. J. Periodont. 14: (Supplement, Jan. 1943) 5 (April) 1943.

6. Linghorne, W. J., and McConnell, D. C. Studies in the regeneration and reattachment of supporting structures of the teeth. I. Soft tissue reattachment. J. D. Res. 29:419 (Aug.) 1950; II. Regeneration of alveolar process. 30:604 (Oct.) 1951.

Fig. 10 • Clinical picture of patient whose roentgenograms are seen in Figure 9. Notice wandering of teeth. Gingivitis is a complicating factor

Fig. 11 • Gingivitis showing edema with gingival enlargement. This results in pericoronal pockets

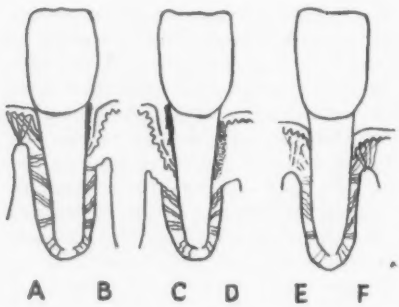


Fig. 12 • Periodontitis and its repair. A. Normal periodontium. B. Calculus in periodontal pocket, apical proliferation of epithelial attachment, ulceration of pocket (sulcus) epithelium, loss of alveolar bone crest and free gingival and alveolar crest fiber bundles. C. Further progression of periodontal pocket, continued bone destruction and loss of horizontal fiber group. D. Picture following proper curettage with blood clot in place of pocket and gingival epithelium proliferating to cover surface of clot. E. Epithelium proliferated to contact cementum. Connective tissue beneath, protected from oral environment, organizing and replacing clot. Alveolar bone crest being stimulated by reforming fiber bundle groups. F. Repaired periodontium with reattached alveolar crest, free gingival and horizontal fiber bundles, some bone regeneration and reattachment of epithelium to cementum. Note that the alveolar bone crest level and free gingival margin are below the level seen in A but no pocket is present. This restoration can occur only under ideal conditions

Whether or not the epithelial attachment fastens itself to the tooth again has also been questioned. It is highly improbable that the epithelium will ever reattach to the enamel, but this is not important clinically. Since the epithelial attachment proliferated onto the cementum from the enamel, it seems likely that new epithelium, covering and repairing a gingival defect, may reattach itself to cementum.

It must be thoroughly understood that there are limitations to the ability of tissues to return to their original state after injury. This is especially true in periodontal disease where the intraoral irritants, trauma and bacteria, are constant threats. Since the possibility exists the extent of regeneration in each case is a challenge to the dentist.

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Weapons of War • In Nature's battle against disease the physician is but the helper who furnishes Nature with weapons, the apothecary is but the smith who forges them. *Paracelsus (1493-1541).*

Local factors in periodontal disease

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Periodontal disease is used as a collective term to designate a complex of pathologic processes in the supporting structures of the teeth. Stomatitis, involving the gingival mucosa, blastomatoid conditions and neoplasms do not exclusively involve the functional unit which constitutes the periodontium, and, therefore, should not be included in periodontal disease. The pulpal-caused periapical diseases usually are listed as separate entities.

The anatomic and physiologic peculiarity of the periodontium makes comparison with other parts or organs of the body difficult. It represents a functional unit which basically is a joint. However, it has an exposed surface which makes it vulnerable to a large number of irritants which ordinarily do not have access to joints. The periodontium, therefore, in addition to dysfunctional irritation common to all joints, is affected by surface irritation and inflammation. Reactions to injuries vary in the periodontium as well as in other joints or organs according to constitutional, nutritional and metabolic status.

The etiology and pathogenesis of a great number of diseases have been clarified by modern cellular pathology, bacteriology and biological chemistry. However, in spite of extensive clinical and histopathologic research, periodontal disease still remains a controversial subject. It has been established clinically that a number of irritants or combinations of irritants are capable of producing periodontal disease. Histopathologic changes provide additional information about the causative factors. But until the effect of of individual as well as various combinations of etiologic factors is well established, it will be impossible to arrive at any definite conclusion about the succession of events which occur in the development of periodontal disease. Periodontal disease involves a combination of dentotechnical and biological problems. The multiplicity of potentially pathogenic factors makes a clinical evaluation of each separate factor difficult. Only a rough differentiation can be made histologically between nonspecific exudative inflammation which follows surface irritation and aseptic degeneration and inflammation which follows circulatory disturbances of dysfunctional origin.

CLINICAL SIGNIFICANCE OF LOCAL FACTORS IN PERIODONTAL DISEASE

The literature on this subject often is of historical rather than informative value because the reports are based on clinical impressions rather than recorded scientific investigations. The detrimental effect of different local irritants is obvious to every practicing dentist but little planned experimental research has been undertaken on the role of local factors in the etiology of periodontal disease. Although local factors generally are supposed to cause 80 to 90 per cent of the

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cases of periodontal disease,[1] most research has been undertaken on the systemic factors.[2]

A number of irritants, according to reports in the literature[3,4] and clinical experience, are considered to be the local causes of periodontal disease. They are as follows:

1. Surface irritants: dental plaques, materia alba, and calculus; bacterial irritation; faulty toothbrushing and massage habits; faulty dentistry; food impaction; (the last-named three may act also as dysfunctional irritants); chemical irritants, and atmospheric irritation.

2. Dysfunction: hyperfunction and hypofunction.

Fauchard[5] stated as early as 1746 that, "The most common cause of the loss of teeth is the negligence of those people who do not clean their teeth when they might, and that they perceive the lodgement of this foreign substance [tartar] which produces disease of the gums." It has been recognized for a long period of time that there is an association between calculus and periodontal disease[6] and this relationship also has been shown experimentally in animals.[7] There has been much discussion, however, as to whether calculus causes periodontal disease or whether calculus is deposited as a result of periodontal disease.[8,9] Although calculus formation always has been a subject for dental research, the complete mechanism of this process has not been explained. A recent article by Hodge and Leung[10] presents a good review of the current theories of calculus formation. The chemical, mechanical, and bacterial irritation produced by materia alba, dental plaques, and calculus is the most common and the most important source of gingival inflammation.[11] The bacteria ordinarily present in the mouth will not invade healthy mucosa, but they participate in the formation of materia alba, dental plaques and calculus.[8,12] On ulcerated surfaces inflammation may be produced by bacteria directly or by their toxins. Furthermore, bacterial toxins, combined with end products of proteolytic fermentation such as hyaluronidase[13] and other enzymes, are active irritants in the production and spread of periodontal inflammation.[14] The production of a local or fixed allergic reaction in the gingival tissues by bacterial toxins and enzymes has been suggested by Hulin[15] and a group composed mainly of French investigators.[16] It has been established by animal experimentation[17-21] and by sur-

1. Orban, B. Periodontal disturbances: practical management. J.A.D.A. 35:545 (Oct. 15) 1947.

2. Held, A. J. The aetiology of parodontal diseases. Internat. D. J. 1:75 (Dec.) 1950.

3. Miller, S. C., and Firestone, J. M. Psychosomatic factors in the etiology of periodontal disease. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 33:675 (Sept.) 1947.

4. Miller, S. C. Textbook of periodontia, ed. 3. Philadelphia, The Blakiston Co., 1950.

5. Fauchard, Pierre. The surgeon dentist or treatise on the teeth. Paris, Jean Mariette, 1746. Translated by Lilian Lindsay. London, Butterworth Co., 1946.

6. Bass, C. C. Relation of the inner border of subgingival calculus to the zone of disintegrating epithelial attachment cuticle. Oral Surg., Oral Med. & Oral Path. 3:1125 (Sept.) 1950.

7. King, J. D. Experimental investigations of parodontal disease. I. The significance of the blood supply in gingival health and disease of the ferret. Brit. D. J. 77:213 (Oct. 20) 1944; 77:245 (Nov. 3) 1944.

8. Box, H. K. Formation of subgingival calculus. J. Canad. D. A. 8:415 (Sept.) 1942.

9. Bunting, R. W., and Hill, T. J. Textbook of oral pathology, ed. 2. Philadelphia, Lea & Febiger, 1940.

10. Hodge, H. C., and Leung, S. W. Calculus formation. J. Periodont. 21:211 (Oct.) 1950.

11. Rosebury, Theodor. Nature and significance of infection in periodontal disease. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 33:658 (Sept.) 1947.

12. Bartels, H. A. Bacterial growth and crystal formation: a possible factor in calculus formation. J. D. Res. 29:436 (Aug.) 1950.

13. Aisenberg, M. S., and Aisenberg, A. D. Hyaluronidase in periodontal disease. Oral Surg., Oral Med. & Oral Path. 4:317 (March) 1951.

14. Fish, E. W. Parodontal disease. London, Eyre and Spottiswoode, Ltd., 1946.

15. Hulin, C. Les paradontoses pyorrheiques. Paris, R. Foulon, 1941.

16. Albanese, P. Le Pyorrhoe Alveolaire, Maladie Allergique. Schweiz. Monatschr. f. Zahnh. 8:844, 1939.

17. Burwasser, P., and Hill, T. J. The effect of hard and soft diets on the gingival tissues of dogs. J. D. Res. 18:389 (Aug.) 1939.

18. King, J. D., and Glover, R. E. Relative effects of dietary constituents and other factors upon calculus formation and gingival disease in ferret. J. Path. & Bact. 57:353 (July) 1945.

19. King, J. D., and Gimson, A. P. Experimental investigations of paradontal disease in the ferret and related lesions in man. Brit. D. J. 83:126 (Sept.) 1947.

20. Rushton, M. A. The epithelial downgrowth on the molar roots of golden hamsters. Brit. D. J. 90:87 (Feb. 20) 1951.

21. Sognnaes, R. F. Experimental production of periodontal disease in animals fed on a purified, nutritionally adequate diet. (Abst.) J. D. Res. 26:475 (Dec.) 1947.

veys of human beings,[22-26] that a soft nondetergent diet increases the accumulation of accretions on the teeth and thereby increases the incidence of periodontal disease. Detergent coarse food or good artificial oral hygiene[27] will increase the protective keratohyaline layer[28, 29] of the gingival mucosa. The only practical way known at present to reduce the harmful plaque and calculus formation is an adequate mechanical oral hygiene.[30]

Sharp particles in the food or faulty toothbrush and massage habits will cause surface injuries and gingival inflammation.[14] Vigorous faulty use of toothbrushes or toothpicks over a long period of time also may cause circulatory disturbances and pressure atrophy of the gingiva.

It is generally recognized that faulty dentistry will cause periodontal disease and numerous condemnations of poor dentistry can be found in the literature.[31] Poorly constructed restorations or appliances may cause surface injury by overhanging margins, by impingement or by promotion of food impaction and increased retention of food, debris and bacteria. Periodontal pockets have been produced experimentally in healthy rhesus monkeys by the use of overhanging margins on metal caps cemented on the teeth.[32] Dysfunctional irritation is another common sequence of inadequate dentistry. A shiny polished gold inlay may have a poor marginal adaptation, an unsatisfactory contact, a flat contour, a faulty marginal ridge or a hyperfunctional occlusal contact.

Food impaction[33] may be the result of poor dentistry, abnormal tooth anatomy, caries, malposition and traumatic occlusion. When food impinges on the gingiva, it causes chemical and bacterial irritation if allowed to decompose, and it also may cause circulatory disturbances by wedging of the teeth.

Surveys have revealed a higher prevalence of periodontal disease in tobacco smokers than in nonsmokers.[34, 35, 36] The irritation from smoking may be both chemical and thermal. Burns from caustic drugs may cause an acute gingivitis. Chemical irritation often is aggravated by an allergic response which may explain severe inflammatory reaction from small dosages which ordinarily would be of no consequence. Such instances usually will appear as stomatitis rather than as gingivitis.

Drying of the mucosa by mouth breathing is one of the most commonly overlooked gingival irritants.[37] This low grade chronic irritation will promote a chronic, often proliferative, gingival inflammation making the papillae look blunt and bulbous if they are uncovered by the lips when the mouth is slightly open.

22. Baarregaard, A. Dental conditions and nutrition among natives in Greenland. Oral Surg., Oral Med. & Oral Path. 2:995 (Aug.) 1949.

23. Haber, G. G. The effect of differences in the quality of bread upon nutrition, and the development of dental caries and tartar in Germany and Switzerland. Brit. D. J. 68:142 (Feb. 15) 1940.

24. Haydak, M. H., and others. Clinical and biochemical study of cow's milk and honey as an essentially exclusive diet for adult humans. Am. J. M. Sc. 207:209 (Feb.) 1944.

25. O'Rourke, J. T. The relation of the physical character of the diet to the health of the periodontal tissues. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 33:687 (Sept.) 1947.

26. Pelzer, R. H. A study of the local oral effect of diet on the periodontal tissues and the gingival capillary structures. J.A.D.A. 27:13 (Jan.) 1940.

27. Robinson, H. B. G., and Kitchin, P. C. Effect of massage with toothbrush on keratinization of gingivae. Oral Surg., Oral Med. & Oral Path. 1:1042 (Nov.) 1948.

28. Orban, B. Hornification of the gums. J.A.D.A. 18:1977 (Nov.) 1930.

29. Weinmann, J. P. Keratinization of the human oral mucosa. J. D. Res. 19:57 (Feb.) 1940.

30. Hine, M. K. Use of the toothbrush in the treatment of periodontitis. J.A.D.A. 41:158 (Aug.) 1950.

31. Thomas, B. O. A. The relationship of operative procedures to the health of the periodontal tissues. J.A.D.A. 39:522 (Nov.) 1949.

32. Ramfjord, S. The effect of systemic disease on the periodontium of rhesus monkeys with reference to poliomyelitis, tuberculosis and alloxan produced diabetes. Experimental periodontal reattachment in rhesus monkeys. Doctoral thesis, University of Michigan, 1950.

33. Hirschfeld, I. Food impaction. J.A.D.A. 17:1504 (Aug.) 1930.

34. Herulf, G. Om det marginala Alveolarbenet hos ungdom i Studiealdern, en rontgenstudie. Svenska Tandl. Tidsk. 43:42, 1950.

35. Pindborg, J. J. Tobacco and gingivitis. J. D. Res. 26:261 (June) 1947.

36. Pindborg, J. J. Tobacco and gingivitis. II. Correlation between consumption of tobacco, ulceromembranous gingivitis and calculus. J. D. Res. 28:460 (Oct.) 1949.

37. Lite, T. Mouth breathing: a contributing factor in the etiology of gingival irritation. J. D. Med. 4:13 (Jan.) 1949.

Dysfunctional irritation is a much more controversial subject than the surface irritants. The role of traumatic occlusion in human periodontal pathology has not been satisfactorily investigated. Box[38] and Stones[39] have produced slight evidence of periodontal disease caused by experimental traumatic occlusion in a few animals. Gottlieb and Orban[40] reported that they have forcefully moved numerous teeth a considerable distance without pocket formation. This has been reported also by a great number of other investigators of orthodontic problems.[41-44] These investigations have confirmed that it is possible to move teeth without pocket formation, but little has been learned about the significance of traumatic occlusion from the reported experiments.

Clinically it has been established by a great number of practicing dentists[45-49] that traumatic occlusion is an important factor in the development of periodontal disease, but the presence of other irritants in the mouth always makes it difficult to evaluate one separate factor.[50-53]

A correct diagnosis of traumatic occlusion also takes more time and training than generally is realized so the condition is often unsatisfactorily diagnosed.

Hypofunction leads to disuse atrophy of the periodontal structures and the lowering of their resistance. Furthermore it predisposes to accumulation of retention products on the teeth which increases the surface irritation to the periodontium.

SIGNIFICANCE OF LOCAL FACTORS IN PERIODONTAL PATHOLOGY

Numerous contributors have added valuable information to Boedecker's[54] and Black's[55] pioneer work on periodontal histology, physiology and pathology. Most of the investigators also have attempted to coordinate etiology, clinical pathology and histopathology into a classification of periodontal disease. Investigations have revealed that the histopathologic changes found in periodontal disease are inflammation, hypertrophy, hyperplasia, circulatory disturbances, degeneration, atrophy, and in some instances, pathologic calcification and pigmentation.

Following extensive investigation of biopsy and autopsy material a few of the investigators[56-61] with a broad train-

38. Box, H. K. Experimental traumatogenic occlusion in sheep. Oral Health 25:9, 1935.

39. Stones, H. H. Experimental investigation into association of traumatic occlusion with parodontal disease. Proc. Roy. Soc. Med., Odont. Sect. Proc. 31:479 (March) 1938.

40. Gottlieb, B., and Orban, B. Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne. Leipzig, Georg Thieme, 1931.

41. Breitner, C. Bone changes resulting from experimental orthodontic treatment. Am. J. Orthodont. & Oral Surg. 26:521 (June) 1940.

42. Lefkowitz, W., and Waugh, L. M. Experimental depression of teeth. Am. J. Orthodont. & Oral Surg. 30:277 (June) 1944.

43. Oppenheim, A. A possibility for physiologic orthodontic movement. Am. J. Orthodont. & Oral Surg. 30:277 (June) 1944.

44. Schwarz, A. M. (Translated by Miller, S. C., and Neustadt, E.) Movement of teeth under traumatic stress. D. Items Interest 52:96 (Feb.) 1930.

45. McCall, J. O. Traumatic occlusion. J.A.D.A. 26:519 (April) 1939.

46. Smith, T. Traumatic occlusion and its correction in the treatment of pyorrhea alveolaris. J. Nat. D. A. 8:971, 1921.

47. Karolyi, M. Beobachtungen über Pyorrhea alveolaris. Oest. Ung. Vierteljsch. Zahn. 17:279, 1901.

48. Stillman, P. R. Traumatic occlusion. J. Nat. D. A. 6:691, 1919.

49. Withycombe, R. M. Without the factor of traumatic occlusion true pyorrhea alveolaris does not eventuate. J. Australia D. S. 6:529, 1926.

50. Leonard, H. J. The occlusal factor in periodontal disease. J. Periodont. 14:12 (Jan.) 1943.

51. Lundquist, G. R. Connective tissue changes associated with variable occlusal stresses. J.A.D.A. & D. Cosmos 24:1577 (Oct.) 1937.

52. Miller, S. C. Periodontist's view of occlusion. J. D. Med. 5:66 (July) 1950.

53. Orban, B. Traumatic occlusion and gum inflammation. J. Periodont. 10:39 (Jan.) 1939.

54. Boedecker, C. F. W. Ueber Pericementium and Pericementitis. Correspondenzblatt f. Zahn. Page 145, 1880.

55. Black, G. V. The periosteum and peridental membranes. Den. Review 1:1, 57, 113, 169, 233, 289, 353, 411, 1886-1887.

56. Häupl, K. Die Chronische Zahnfleischentzundung and Zahnlocherung. Berichte von IX Internationalen Zahnärztekongress der F.D.I. Wien und Berlin, Urban und Schwarzenberg, 1936.

57. Häupl, K., and Lang, F. J. Die marginale Paradentitis. Berlin, Hermann Meusser, 1927.

58. James, W. W., and Counsell, A. A histological investigation into "so-called pyorrhea alveolaris." Brit. D. J. 48:1237, 1927.

59. James, W. W., and Counsell, A. The primary lesion in "so-called pyorrhea alveolaris." Brit. D. J. 49:1129, 1928.

60. Bernier, J. L. The role of inflammation in periodontal disease. Oral Surg., Oral Med. & Oral Path. 2:583 (May) 1949.

61. Bernier, J. L. The histologic changes of the gingival tissues in health and periodontal disease. Oral Surg., Oral Med. & Oral Path. 3:1194 (Sept.) 1950.

ing in general and oral pathology claim that all periodontal disease is of inflammatory nature. Another much larger group of well recognized investigators[62, 54, 63-73] maintain that they have observed two main types of periodontal disease—one type caused by local irritants and manifested as inflammation and the other type caused by systemic disturbances and manifested as degeneration. The investigators in this latter group usually list periodontal traumatism as a pathologic entity of its own.[74]

RESPONSE TO SURFACE INJURY

An exudative nonspecific inflammation following surface injury is so commonly observed in the gingival tissues that a slight degree of this inflammation has been considered normal for an adult person by some investigators.[61] That this simple gingivitis is a response to surface injury is generally accepted. This lesion corresponds to a localized dermatitis[75] following localized skin injury, while a stomatitis often corresponds to a more widespread dermatosis. Localized gingival inflammation or gingivitis has never been demonstrated without the presence of local irritants.[56] The amount or degree of irritation which is required to interrupt the normal metabolic process in the cell organization and produce inflammation is dependent on local and general resistance. The threshold for pathologic injury may be so low in some individuals, because of constitutional factors or general metabolic and hormonal disturbances, that the negligible irritant which initiated the inflammatory reaction is difficult to detect clinically.

A hyperplastic proliferative inflammatory response to injury[76, 77] is not peculiar to the periodontium but is seen elsewhere in the body, especially in young individuals with a high potentiality for repair.

A surface inflammation in the gingiva is of great significance because it interferes with the normal function of the gingival tissues and has a tendency to spread into the deeper periodontal structures. This perivascular extension of periodontal inflammation was first called to the attention of dentists by Boedecker[54] in 1880, and was later investigated by Box,[78] Häupl and Lang[57] and Weinmann.[79]

Downgrowth and degeneration of the epithelial attachment follows the inflammatory destruction of the collagen fibers and is caused by stimulation from toxic products in the inflamed area.[80, 81, 82]

62. Hunter, J. A practical treatise on the diseases of the teeth. London, J. Johnson, 1778.

63. Znamensky, N. N. Alveolar pyorrhoea—its pathological anatomy and its radical treatment. Brit. D. J. 23:585, 1902.

64. Hopewell-Smith, A. Pyorrhea alveolaris—its pathohistology, Parts I and II. D. Cosmos 53:397 (April) 1911; 53:981 (Sept.) 1911.

65. Gottlieb, B. Tissue changes in pyorrhea. J.A.D.A. 14:2178 (Dec.) 1927.

66. Gottlieb, B., and Orban, B. Zahnfleischentzündung and Zahnlocherung. Berlin, Berlinsche Veralgsanstalt, 1936, p. 161.

67. Kronfeld, R. The progress of microscopic research in periodontal diseases (Abst.) J. D. Res. 13: 167 (April) 1933.

68. Orban, B., and Weinmann, J. P. Diffuse atrophy of the alveolar bone (periodontosis). J. Periodont. 13: 31 (Jan.) 1942.

69. Becks, H. Normal and pathologic pocket formation. J.A.D.A. 16:2167 (Dec.) 1929.

70. Thoma, K. H., and Goldman, H. M. Classification and histopathology of paradontal disease. J.A.D.A. 24:1915 (Dec.) 1937.

71. Thoma, K. H., and Goldman, H. M. Wandering and elongation of teeth and pocket formation in parodontosis. J.A.D.A. 27:335 (March) 1940.

72. Muller, O. Pathohistologie der Zähne. Basel, Benno Schwabe & Co., 1948.

73. Held, A. J., and Nally, J. N. De la gingivite et la paradentite marginale. Schweiz. Monatsch. f. Zahnh. 58:187, 1948.

74. Nomenclature in periodontia. (Advance report of the Nomenclature Committee of the American Academy of Periodontology, 1947.) J. Periodont. 18: 112 (April) 1947.

75. Ormsby, O. S., and Montgomery, H. Diseases of the skin, ed. 7. Philadelphia, Lea & Febiger, 1948.

76. Coolidge, E. D. Hypertrophic gingivitis. J.A.D.A. 28:1381 (Sept.) 1941.

77. Glickman, I. A basic classification of "gingival enlargement." J. Periodont. 21:131 (July) 1950.

78. Box, H. K. Studies in periodontal pathology. Bulletin no. 7. Toronto, Canadian Dental Research Foundation, May, 1924.

79. Weinmann, J. P. Progress of gingival inflammation into the supporting structures of the teeth. J. Periodont. 12:71 (July) 1941.

80. Fish, E. W. Surgical pathology of the mouth. London, Sir Isaac Pitman & Sons, 1948.

81. Goldman, H. M. Marginal periodontitis. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 31:93 (Feb.) 1945.

82. Häupl, K. Grundriss der Histo-Pathologie des Zahnes und seines Stützapparates. Leipzig, J. A. Barth, 1940.

That resorption of alveolar bone and alveolar process may occur as a sequel of gingival inflammation also is beyond dispute. This review presents a brief outline of the inflammatory periodontal changes caused by surface irritation. Many investigators consider this exudative inflammation the only type of inflammatory reaction in the periodontium, and the only type of periodontal disease which is caused by local injury.

RESPONSE TO DYSFUNCTIONAL IRRITATION

Even minor injury to tissue cells releases cytoplasmic substances which cause dilatation and increased permeability of the adjacent capillaries, and edema from transudation of plasma into the tissue spaces. Slight injury to the endothelial cells lining the vessel walls may cause subtle cellular changes manifested by a roughened surface which may promote adherence of platelets, agglutination, clotting and eventually thrombosis.[83]

If the mechanical injury is of sufficient intensity to disrupt the walls of blood vessels an internal hemorrhage occurs, but in instances of damage to small vessels vasoconstriction is a more important mechanism than thrombosis in the production of stasis. Accumulation of waste products from the overburdened connective tissue cells also has an irritating effect on the capillary walls. This leads to transudation and edema which, in the case of lasting irritation and increased vascular alteration, will develop into exudation and inflammation.

Mechanical disruption of living cells, or thrombosis and internal hemorrhage, is followed by a series of secondary local reactive changes called aseptic inflammation.[83, 84] The effect of dysfunctional circulatory disturbances depends on the extent of the damage and the anatomic, metabolic and functional character of the injured tissues. Ischemia, hyperemia, edema, atrophy, degeneration, and even necrosis may occur. In the periodontium, an area of aseptic inflammation following dysfunctional circulatory disturbances will undergo resolution, and granulation tissue will be organized into functional periodontal structures if given functional stimulus, but the process of aseptic inflammation will continue if the dysfunctional irritation continues.

Many investigators have described the dramatic tissue changes in severe acute traumatic occlusion.[40, 85-91] Thrombosis, necrosis, hyalinization, resorption of bone and cementum, and calcific deposits in necrobiotic and necrotic areas, have been listed as pathognomonic of a specific pathologic entity called "traumatism." The less spectacular tissue changes, with chronic circulatory disturbances of a dysfunctional nature and the healing stages of aseptic inflammation following traumatic injury, have been made another pathologic entity and called "diffuse alveolar atrophy" or "periodontosis." A somewhat similar situation existed in the use of the term "arthrosis." Before different types of arthritis were recognized, and when little was known about the etiology, arthrosis was used as a collective term to indicate the degenerative nature of the disease. Today one would rather use the

83. Anderson, W. A. D. Pathology. St. Louis, C. V. Mosby Co., 1948.

84. Wolf, Elizabeth P. Experimental studies on inflammation. J. Exper. Med. 37:511, 1923.

85. Brauer, W. Ueber traumatische Schädigungen des Zementmantels der Zähne mit einem Beitrag zur Biologie des Zementes. Deutsche Monatsch. f. Zahnh. 45: 769, 1927.

86. Erikson, B. E., Kaplan, H., and Aisenberg M. S. Orthodontics and transeptal fibers. Am. J. Orthodont. & Oral Surg. 31:1 (Jan.) 1945.

87. Coolidge, E. D. Traumatic and functional injuries occurring in supporting tissues of human teeth. J.A.D.A. & D. Cosmos 25:343 (March) 1938.

88. Kronfeld, R., and Weinmann, J. Traumatic changes in the periodontal tissues of deciduous teeth. J. D. Res. 18:441 (Oct.) 1940.

89. Orban, B. Tissue changes in traumatic occlusion. J.A.D.A. 15:2090 (Nov.) 1928.

90. Orban, B., and Weinmann, J. Signs of traumatic occlusion in average human jaws (Abst.) J. D. Res. 13:216 (June) 1933.

91. Stones, H. H. An histological study of the investing tissues of the teeth, the dentine and dental pulp. Brit. D. J. 65:737 (Dec. 15) 1938.

term "traumatic osteoarthritis" than the less descriptive term "arthrosis" if the morbid condition is of traumatic origin.

The concept of diffuse alveolar atrophy, or periodontosis, is founded on autopsy findings of loose vascular connective tissue in the periodontal space, proliferation of epithelial rests and resorption of alveolar bone.[80, 92, 93, 94] This histologic finding is supposed to correspond clinically to migration of teeth and sudden pocket formation without appreciable irritation or inflammation. The autopsied cases had died from systemic diseases which were considered capable of producing periodontal degeneration and, because of lack of adequate control material, an unfounded relationship has been reported to exist between the periodontal changes and systemic diseases.

Recently an investigation of the relation between systemic disease and periodontal state of health was undertaken in the University of Michigan[82] utilizing 46 rhesus monkeys. Seven of the animals were kept as controls, 10 animals suffered from severe human tuberculosis, 19 were inoculated with poliomyelitis virus and developed acute poliomyelitis with high fever. Finally, in 10 animals an attempt was made to produce alloxan diabetes. In some of the animals slightly too high occlusal amalgam fillings were inserted. In animals that were shedding their teeth, the periodontal tissues around the first permanent molars as well as the tissues around deciduous teeth with short remaining roots and around the teeth with the high fillings showed the well established histologic signs of traumatism (hemorrhage, thrombosis, degeneration, necrosis, bone and sometimes cemental resorption). Furthermore, loose vascular connective tissue and extensive resorption of alveolar bone were observed around overburdened teeth. These areas resembled the histologic appearance of periodontosis[86] so closely that the traumatic nature of the lesions could only be established following the examination of similar lesions in the control animals and by a further review of material from human beings. No clinical or histologic difference was observed between the periodontal tissues and collagen tissues from joints in the sick monkeys when compared with the healthy control animals. These findings substantiated what Häupl[56] and Mezl[95] have claimed about the histologic appearance of the so-called "periodontosis" as a manifestation of dysfunctional irritation.

The difference in manifestations and development of the various pathologic processes in the periodontium can be understood only after a clear concept of the periodontal blood supply is acquired. The well established "textbook pattern"[4] of periodontal[79] blood supply has been over-emphasized and the individual anatomic variations have been overlooked. This accounts for a tendency toward generalization which anatomically is not justified. The periodontal membrane often gets its blood supply through haversian canals penetrating the alveolar bone, and the supra-alveolar soft tissues get their blood supply through interdental vessels penetrating the alveolar crest. Dysfunctional circulatory disturbances then will affect the periodontal membrane primarily and secondarily the alveolar bone and cementum.[96] The degeneration and aseptic inflammation will be confined principally to the part of the periodontium which is situated apically to the margin of the alveolar crest, and the traumatism, at least in the early stages, will be of little significance to

92. Gottlieb, B. Zur Aetiologie und Therapie der Alveolarpyorrhoe. Zeitsch. Stomat. 18:59, 1920.

93. Gottlieb, B. Die Diffuse Atrophie des Alveolarknochens. Zeitsch. Stomat. 21:195, 1923.

94. Orban, B. Histopathology of periodontal diseases. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 33:637 (Sept.) 1947.

95. Mezl, Z. Contribution a l'histologie pathologique du paradentium. Paradentologie 2:60, 1948.

96. Henry, J. L., and Weinmann, J. P. The pattern of resorption and repair of human cementum. J.A.D.A. 42:270 (March) 1951.

the gingiva. But commonly blood vessels enter the periodontal membrane and pass into the gingiva over the margin of the alveolar crest which can be demonstrated both before and following resorption of alveolar bone and alveolar process. A horizontal overload on such a tooth would inflict damage on vessels which partly supply the gingiva. It should also be emphasized that stretching or distention has practically the same effect on the tissues as compression (Forbus),[97] so that the traumatic effect is not limited to the side of compression. An interference with the gingival blood supply will lead to edema and all the other sequela of circulatory disturbances listed previously. The lowering of resistance which accompanies the metabolic disturbances in such region is of great significance in the gingiva because it is continuously exposed to surface irritants. Severe traumatic injury, causing thrombosis of vessels which supply large portions of the periodontal tissues and extending to the epithelial attachment, will predispose to sudden and rapid pocket formation.

The periodontal necrotic foci[14, 98, 99, 100] which have received great attention lately may be the sequence of exudative marginal inflammation (cf. Menkin's "necrosin")[101] or they may be necrotic areas in the aseptic traumatic inflammation which follows dysfunctional irritation. Similar changes are seen, for example, in traumatic temporomandibular joint arthritis.

Another sequence of circulatory disturbances in the periodontium is gingival and periodontal atrophy. Atrophy is caused by some change or disturbance in the metabolism of the involved cells, mainly because of interference with the nutrition of the cell. Circulatory disturbances caused by pressure, inactivity, over-activity, arteriosclerosis, injury to the nerve supply of an organ, hormonal imbalance, inadequate nutrition, are all factors which may alter the cell metabolism and cause atrophy. However, if the atrophy is confined to a single part of the body which is not undergoing hormonal involution the logical explanation of the atrophy is a local cause. Gingival atrophy may be a pressure atrophy from faulty use of mechanical aids in oral hygiene, or it may be a sequence of exudative inflammation caused by other surface injuries. Probably the most common source of periodontal atrophy is circulatory disturbances from dysfunction. In cases of traumatic occlusion where good oral hygiene is practiced, a gingival recession and thickening of the gingival margin may be the only clinical sign of traumatism.

Calcified deposits are not unusual in the periodontal tissues,[102] and they are of the same significance as in an inflammatory process in an area of connective tissue elsewhere in the body. Foreign bodies, such as amalgam particles, causing pigmentation, may be present locally, or be borne by the blood stream as is bismuth. They act as local irritants and produce inflammatory reaction.

A wide variation in response to local injury has been observed in the periodontium as well as in other parts of the body, but no type of periodontal disease has been found to be related to a definite systemic disturbance.[103] The periodontal metabolism is dependent on adequate nutritional supply[104-107] and normal hormonal activities.[108] Being

97. Forbus, W. D. Reaction to injury. Baltimore, Williams & Wilkins Co., 1943.

98. Box, H. K. Concerning the pathogenesis of periodontal disease. J. Periodont. 19:11 (Jan.) 1948.

99. Ray, H. G., and Orban, B. Deep necrotic foci in the gingiva. J. Periodont. 19:91 (July) 1948.

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continuously exposed to local injuries, a lowering of the general tissue metabolism and resistance may manifest itself early in active periodontal disease. In modern man the periodontium is a vulnerable organ because it does not receive proper functional stimulus during its development and daily use. Everything that will improve general health, nutrition and metabolism is of value in building up resistance against injury to the periodontal tissues as well as in increasing the resistance against tubercle bacilli and other irritants elsewhere in the body.

The tissue changes found in the common types of periodontal disease, including periodontosis and atrophy, can be explained on the basis of reaction to local injury. An unjustified "systemic-factor escape" from responsibility for poor management of local irritants, especially of a dysfunctional nature, has delayed the progress of dentistry and the treatment of periodontal disease and has resulted in the unnecessary loss of a tremendous number of teeth. It is time to utilize the well established facts of etiology to improve therapeutic procedures in periodontics.

SUMMARY

1. The clinical and histological reaction to various types of local periodontal injury has been discussed.

2. The exudative periodontal inflammation caused by surface injury has been distinguished from an aseptic periodontal inflammation related to circulatory disturbances and caused mainly by traumatic occlusion.

3. "Periodontosis" has been explained on the basis of local circulatory disturbances of dysfunctional nature.

4. All common types of periodontal disease, including atrophy and periodontosis, can be caused by local injury.

5. The constitutional state of the individual, including nutrition, hormonal balance and metabolism, is always an important factor in the development of periodontal disease, because it determines the response to the local injuries which, in many instances, initiate the disease.

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The Case Report • After all, the only really valid reason for writing a scientific medical article is to present the results of useful investigation or seasoned experience, thus adding to the sum of medical knowledge, or to bring together and correlate existing knowledge in order to make it more easily available. Only occasionally is a single case worth reporting, to remind a journal's readers of the existence of some condition that may cause diagnostic confusion or to add to the knowledge of its treatment; it should be reported with the utmost brevity. A case report should always point a moral, whether or not it may adorn a tale. *New England Journal of Medicine 245:988 December 20, 1951.*

The systemic factors in periodontal disease

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Procedures that have been employed to ascertain the possible systemic factors in periodontal disease involving loss of alveolar bone include blood and urinary analyses, dietary studies, estimation of the basal metabolic rate and specific dynamic action, evaluation of hormone activity and physical examinations.

BLOOD STUDIES

Regarding the blood studies, several investigators have reported higher than normal values for total calcium, total cholesterol, uric acid, sugar and phosphatase (in gingival blood) in various percentages of their cases. Lower than normal values have been reported for vitamin C, erythrocyte count and carbon dioxide combining power. Other workers have failed to confirm the findings for calcium, uric acid, sugar and cholesterol. In view of the uncertainty of these results, we[1-4] carried out a comprehensive blood study in 229 cases, divided into four groups. There were 54 cases in the control group which consisted of individuals free from periodontal disease; 77 cases in the periodontosis group (Group A); 59 cases in a more advanced periodontosis group (Group B); and 39 advanced cases of periodontitis. The mean ages were 33.5, 27.3, 39.1 and 48.8, respectively.

Group A,[5] predominantly females, included either young individuals whose roentgenograms showed signs of severe alveolar bone loss, generally in specific areas, or patients in whose mouths local factors were not prominent and apparently did not account for the severe loss of alveolar bone. As a rule, the gingivae appeared pink and stippled but there was slight erythema of the interdental papillae in several areas in a majority of the cases. Little or no calculus was present. A diagnostic sign was the migration of the maxillary or mandibular incisors with resulting diastemata. Roentgenographic examination revealed loss of alveolar bone in the first molar and incisor regions. In many regions there was a radiolucency of the alveolar crest and a widening of the periodontal membrane. More alveolar bone was present about the bicuspid teeth in both arches, especially the mandible. These observations were rather constant

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in this group in individuals under 25 years af age.

Group B, also predominantly females, consisted of individuals in whom one or more local factors were more prominent than in Group A. As a rule, the patients in this group were older than those in Group A. The gingivae were red and the papillae had a purplish tinge. No stippling was present and the papillae had lost their firmness and appeared glossy and spongy. Many of the teeth showed second and third degree mobility. Roentgenograms of this group showed an almost complete breakdown of the alveolar process.

The periodontitis group, approximately equally divided between males and females, consisted of individuals with heavy deposits of calculus. The gingivae were inflamed, hypertrophied, purplish in color and bled easily. In many cases pus oozed from the gingival crevice upon finger pressure. There was little or no elongation or protrusion of the anterior teeth. As a rule, the teeth showed mobility only in the advanced stages. Roentgenographic examination revealed a horizontal loss of alveolar bone which was severe in a majority of the cases.

The results of the blood analyses are given in Table 1. Differences were found between the control and each of the periodontal groups for the following: vitamin C, glucose, globulin, albumin, calcium-ion, alkaline phosphatase, red blood cell sedimentation rate, red blood count, white blood count and neutrophils. However, the deviations of individual values from the normal ranges were in general small. There were no fundamental differences between the three periodontal groups.

The greater percentage of deviations from the normal blood ranges found in the periodontal groups and the small extent of individual deviations suggest that one or more systemic conditions, mainly of a subclinical nature, are associated with these diseases in many of the subjects studied. Since deviations from the normal ranges were obtained for several apparently unrelated blood constituents, it does not appear that a com-

Table 1 • Percentage of cases with values outside the normal range in the control and experimental groups

Blood constituents studied	Control group		Periodontosis group (group A)		Periodontosis group (group B)		Periodontitis group	
	Above	Below	Above	Below	Above	Below	Above	Below
Vitamin C_s in plasma		15.0		26.3		42.5		41.0
Glucose in whole blood	1.8		16.1		15.1		22.5	
Globulin in serum	3.2		28.8		20.6		28.6	
Albumin in serum		3.2		14.9		10.3		21.4
Calcium-ion in serum		5.7		13.5		12.1		8.1
Alkaline phosphatase in serum	0.0		8.6		13.8		20.0	
Red blood cell sedimentation rate	11.5		42.5		41.5		50.0	
Red blood count	3.1	3.1	18.8	10.7	6.7	16.6	11.8	17.7
White blood count	0.0		14.7		6.5		11.3	
Neutrophils	0.0		13.4		9.7		11.5	11.5

mon systemic condition operates as a modifying or predisposing factor in these conditions. Furthermore, the nature of a systemic condition that may be involved cannot be definitely stated on the basis of these data. The general similarity of the results in the three periodontal groups is of interest in view of the emphasis that is generally placed on local factors in the mouth as the major, if not the sole, cause of periodontitis. It should be recalled that in contrast to this form of periodontal disease, periodontosis may occur in individuals in whose mouths local factors are either absent or not prominent.

For the other blood constituent studies, deviations from the normal ranges were obtained for the control and experimental groups, but there were no differences between these groups. This was the case for total calcium, inorganic phosphate, sodium, chloride, and also for total cholesterol, free cholesterol, nonprotein nitrogen, urea, uric acid, carbon dioxide capacity, hemoglobin, vitamin A and carotene.

Of interest in connection with the blood studies are the findings of Seidler, Miller and Wolf[6] in glucose tolerance tests in cases of advanced alveolar bone destruction in young individuals (mean age 23.5). Glucose tolerance curves, obtained by administering 100 Gm. of glucose, were found to be flattened in 34 of 35 cases. The average blood sugar values obtained were: fasting, 85.1 mg. per cent; one hour, 99.1 mg. per cent; two hours, 89.1 mg. per cent; three hours, 85.6 mg. per cent. Normally, according to these investigators, the blood sugar would be about 130 mg. after the first hour.

Eight cases showed a "reverse" curve where the administration of glucose resulted in a fall rather than an increase of blood sugar concentration. The reason for this result has not been definitely established. It is known that dextrose tolerance tests vary definitely with the character of the antecedent diet. For example, the rise in blood sugar in this test will be distinctly less if the subjects are on a high carbohydrate diet. Seidler and co-workers reported that 80 per cent of their subjects consumed what appeared to be excessive amounts of carbohydrate, so that the flattened curve may be secondary to a high carbohydrate diet.

More recently Ostrom, Switzer and Fosdick[7] determined the glucose tolerance in a group of 41 individuals, average age 40, suffering from periodontal disease characterized as advanced periodontitis. A comparison was made with a group of 16 individuals, average age 26, having optimum gingival health. It was reported that although the fasting blood sugar of the periodontitis cases was within the normal range, the blood sugar levels following the injection of 50 cc. of 50 per cent glucose rose higher and persisted longer than those of the normal individuals. In the periodontitis group the half-hour peak was 164.5 mg. per hundred cubic centimeters of blood and the duration of hyperglycemia was 1.75 hours as compared with a half-hour peak of 127.5 mg. and duration of hyperglycemia of 1.10 hours in the control group. It is clear that the responses to the administration of glucose were greatly different in these two investigations. This may be owing to a difference in the type of cases studied.

DIETARY STUDIES

Several dietary studies have been made in an effort to throw light on possible systemic factors in periodontal disease. In our[4] investigation a record was made of the food consumed during a one week

6. Seidler, B.; Miller, S. C., and Wolf, W. Systemic aspects of precocious advanced alveolar bone destruction: preliminary report. J.A.D.A. 40:49 (Jan.) 1950.

7. Ostrom, C. A.; Switzer, J. L., and Fosdick, L. S. Chemical studies in periodontal disease. VII: Correlation of blood glucose tolerance and tissue glycogen concentration in periodontal disease. J.A.D.A. 42:388 (April) 1951.

period for 22 control and 24 periodontosis cases. From this information the intake of some of the essential food constituents was calculated (Table 2). On the basis of the daily dietary allowances recommended by the Food and Nutrition Board of the National Research Council,[8] the caloric intake was low in 45 per cent of the control group and in 48 per cent of the periodontosis group. Results of a similar nature were found for protein, calcium, phosphorus, iron, thiamin, riboflavin and niacin. In general, there was no fundamental difference between the two groups. It should be noted that the recommended daily allowances are admittedly higher than average requirements, and that the deviations from these allowances were, in general, small.

In contrast to these results, it was found that with few exceptions, vitamins A and C were ingested in adequate amounts by both groups. The fact that plasma vitamin C was low in a large percentage of the periodontosis cases may mean that there was some interference in the utilization of this vitamin. There was no evidence in this study of excess carbohydrate intake by the periodontosis group as reported by Seidler and co-workers for their patients with precocious advanced alveolar bone atrophy.

As a check on the dietary studies urinary determinations were made to test for the possibility of a subclinical deficiency of thiamin, riboflavin and nicotinic acid. These tests failed to reveal such a deficiency in patients in either the periodontosis or the periodontitis groups. Furthermore when the approximate amounts of these substances in the urine were compared no differences were noted between the control and peridontal groups.

The early dietary studies of Tishler[9] of 101 periodontal disease cases between ages of 18 and 63 are of interest. The lesions of these patients varied from an incipient to an advanced type of periodontal disease with or without visible suppuration. Based on the food eaten during a one week period, low intakes were found as follows: protein in 47 cases, calcium in 62 cases, iron in 43 cases, vitamin A in 60 cases, vitamin B in 41 cases, vitamin C in 34 cases.

Table 2 • Percentage of cases showing low food intake*

Essential food constituents	Control group (per cent)	Periodontosis group (per cent)
Calories	45	48
Protein	23	24
Calcium	73	70
Phosphorus	55	58
Iron	36	45
Thiamin	36	21
Riboflavin	45	50
Niacin	41	41

*Based on the dietary allowances recommended by the Food and Nutrition Board, National Research Council.

Radusch[10] also reported a reduced intake of calcium in various stages of periodontal disease on the basis of a dietary survey over a period of one week. Furthermore she reported a significant association between calcium intake and degree of periodontal disease, with lower intakes in the severer cases. Her results for phosphorus were not as consistent, but the data of the experimental groups indicated a significant correlation for this factor as well as for protein. Little or no association was reported for vitamin A, vitamin C, iron or acid-base excess and the extent of periodontal condition.

ALTERED CAPILLARY PERMEABILITY

The possibility that altered capillary permeability may be a contributing factor

8. Food and Nutrition Board, National Research Council. Recommended dietary allowances. Reprint and Circular Series, Number 129, October, 1948.

9. Tishler, B. Diet analysis of one hundred and one periodontoclasia cases. J.A.D.A. 16:1037 (June) 1929.

10. Radusch, D. F. The relationship between periodontal condition and certain dietary factors. J.D. Res. 18:305 (Aug.) 1939.

Table 3 • Summary of results on metabolic changes in periodontosis

Author	Basal metabolic rate				Specific dynamic action			
	Number of cases	Lowered	Normal	Increased	Number of cases	Lowered	Normal	Increased
Boenheim	161	34	81	46	161	86	66	9
Cahen	6	1	7	—	8	6	2	—
Citron	81	7	51	23	70	37	33	—
Herz	6	5	1	—	—	—	—	—
Lebermann	49	2	45	2	20	6	14	—
Nothmann	21	—	16	5	—	—	—	—
Weinmann	23	7	11	5	23	21	2	—
Total	347	56	212	81	282	156	117	9

in periodontal disease has been presented by Burket and Kreshover,[11] who found that a large percentage of patients with this disease had an abnormally high petechial count—that is, increased capillary fragility—as compared to a small percentage in a control group of the same age range. There was no correlation between the petechial count and blood ascorbic levels before or after saturation of the patient with ascorbic acid. They, therefore, suggested that the abnormal capillary fragility findings may be an indication, among other things, of a deficiency of vitamin P.

ENDOCRINE GLAND STUDIES

Since an increase or decrease in the activity of the endocrine glands may be an etiologic factor in periodontosis, one of the studies we carried out was the determination of the neutral 17-ketosteroids in urine. These substances are excretion products of some of the steroids of the adrenal cortex, while some of them originate in part from the male gonads. The determination of the 17-ketosteroids in urine is, therefore, essentially a measure of adrenocortical and male gonadal activity. It has been shown that these glands are related to the activity of the osteoblasts.

The results obtained for the females in this study were as follows: there were 20 cases in the control group with a mean of 11.3 mg. per 24 hours; the periodontosis group consisted of 33 cases with a mean of 9.8 mg. per 24 hours. The difference between the means divided by the standard error is 1.44, which indicates that there is no significant difference between the groups. However, it should be noted that 30 per cent of the cases in the control group gave values below 9.3 mg. per 24 hours, as compared with 52 per cent in the periodontosis group. The value 9.3 is regarded as the mean for females between the ages of 17 and 64.

Inasmuch as periodontosis is a disease that occurs predominantly in females of reproductive age, especially during adolescence and early maturity, we[12] carried out studies which might throw light on ovarian function in patients with this disease. This was done by determining the estrogen content of urine, and by serial vaginal smears and basal body temperature recordings. For the estrogen determination, 24 hour urine specimens were collected, generally on the seventh day of the menstrual cycle, and on the day on which there was a noticeable rise in temperature. Vaginal smears were made on alternate days during a complete cycle beginning with the first day.

11. Burket, L. W. Oral medicine. Philadelphia, J. B. Lippincott, 1946, p. 411.

12. Karshan, M.; Wiener, R., and Tenenbaum, B. Studies of ovarian function in periodontosis. J.D. Res. 30:460 (Aug.) 1951.

The indexes employed to determine the quality of the cycle were the degree of cornification, evolution of cell type and temperature.

Our preliminary results showed a wide range of ovarian activity among the 20 patients studied. Two patients were considered to have anovulatory cycles. The quality of the cycles in the ovulatory cases was poor in 2 cases, fair in 6, good in 8, and excellent in 2. With two exceptions, the estrogen output was within the normal limits. However, on the basis of the smear picture and temperature recordings, it may be said that half of the patients studied showed some impairment in ovarian function.

Several reports have appeared on the basal metabolic rate and specific dynamic action in periodontal disease. It is known that in most instances deviations from normal metabolic rates are due to hyperfunction or hypofunction of the thyroid gland. Specific dynamic action is a measure of the increase in basal metabolism after the ingestion of a test meal. The normal increase in metabolic rate after a test meal of protein is approximately 20 per cent. The significance of deviations from the normal specific dynamic action is less certain than deviations in the basal metabolic rate.

A summary of the early findings in this field, as compiled by Boenheim,[13] is shown in Table 3. More recently, Seidler, Miller and Wolf[6] reported somewhat lowered basal metabolic rates and specific dynamic action in patients with precocious advanced alveolar atrophy. In 29 of 35 cases, the basal metabolic rate was below zero per cent, the mean of the 35 cases being —6.1 per cent. The average specific dynamic action obtained by repeating the basal metabolism test one and a half hours after a high protein meal was 6.7 per cent as compared with a normal rise of about 16 per cent on the test meal. Our own basal metabolism studies did not reveal a difference between the control and periodontal disease groups.

INCIDENCE OF SYSTEMIC DISEASE

In a further effort to ascertain the possible systemic factors in periodontal disease, physical examinations were made and case histories taken of 50 of our periodontosis cases from 14 to 46 years of age. Twenty-one gave essentially negative results. In 29 cases a number of apparently unrelated conditions were found as follows: systolic murmur (or mitral stenosis) in 6 cases; diabetes in 3

13. Boenheim, F. Pathogenic importance of the endocrine glands in perodontal disease. J.D. Res. 17:19 (Feb.) 1938.

Table 4 • Incidence of systemic disease in 200 individuals without periodontosis and 200 individuals with periodontosis

Systemic disease	With periodontosis (cases)	Without periodontosis (cases)
Diabetes mellitus	9	1
Syphilis	8	1
Gastrointestinal ulcer	20	9
Cancer	8	0
Arthritis	13	36
Cholecystitis	7	18
Cardiac disease	4	7
Respiratory disease	11	10
Hypertension	7	9
Goiter	9	7

cases; hay fever in 3 cases; mild hyperthyroidism in 2 cases; intermittent dyspepsia in 2 cases; seborrheic dermatitis in 2 cases The following were found, each in one case: enlarged liver and tender spleen; amoebic colitis; recurring peritoneal abscesses; hysterectomy because of fibroid tumor; recurring maxillary sinusitis; Hodgkin's disease (chronic inflammatory enlargement of the lymph nodes, enlargement of spleen and often of liver and kidneys); cervical lymph node tuberculosis; history of syphilis and gonorrhea; cystocele (hernia of the bladder) and palpable right kidney; thickening and roughening of the skin and moderate hirsutism. It is thus clear that these examinations did not show a consistent pattern in the periodontosis patients.

Working along similar lines, Goldstein[14] reported on the incidence of systemic disease in groups of patients with and without periodontal disease. The mean age of the control and periodontal groups were 42.2 and 51.6, respectively. Results were negative in approximately half the cases in each group. Systemic conditions which were found in the remainder of the cases in each group are summarized in Table 4. Diabetes, syphilis, gastrointestinal ulcers and cancer were more frequent in the individuals with periodontosis, whereas arthritis and cholecystitis were present more frequently in the individuals without periodontosis. As in our study, the findings of Goldstein and those of others do not point to a specific systemic condition associated with periodontal disease.

While these observations point out that a large number of systemic conditions are found in coexistence with periodontal disease, in the main no definite conclusion can be drawn as to cause and effect. In some instances the causal relationship seems to be more apparent. In diabetes,[15] for example, the disturbance in metabolism and the decrease in resistance to infection may result in gingival inflammation and destruction of alveolar bone. It may well be, as suggested by Bunting, that "disturbances of the general health and nutrition of the body frequently produce marked changes in the tone and vital resistance of the gingival and periodontal tissues and render them more susceptible to injury from traumatic stresses and bacterial invasion." In other words, systemic factors may reduce the resistance of the tissue, making them more susceptible to local factors which may produce periodontal disease. It is more difficult to account for the great destruction of alveolar bone in individuals in apparent good health and in whom local conditions assumed to be related to periodontal disease are either absent or not prominent.

14. Goldstein, H. Systemic and blood findings in parodontosis. J.D. Res. 17:15 (Feb.) 1938.

15. Rutledge, C. E. Oral and roentgenographic aspects of the teeth and jaws of juvenile diabetics. J.A.D.A. 27:1740 (Nov.) 1940.

Health and Fulfillment • A nation can reach pre-eminence and fulfill its destiny only if its people are capable of high productivity and this, in turn, is possible only when the people enjoy good health. Medicine has aided greatly in promoting the national health; one evidence of this fact is the pronounced increase in the life span. It must be recognized, however, that still too frequently life is prolonged into old age without health and happiness, a circumstance directing attention to the common chronic diseases—hypertension, diabetes, atherosclerosis, cancer, arthritis, degenerative diseases of the liver and kidney—whose etiology is not well defined and which continue as a major challenge to the medical profession and the health agencies. *"Nutrition in Chronic Disease," Nutrition Reviews 10:38, February, 1952.*

Gingivitis

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The investing tissues of teeth have a histologic and physical character and form suited to the functional use of the teeth. As a consequence, these tissues are subject to diseases, many of which are unlike those of other tissues of the body. This fact, together with the manipulations required in treatment, has made dentistry a highly specialized professional field.

ETIOLOGIC FACTORS

The different diseases of the gingiva and the investing tissues of the teeth should be considered separately as to both cause and treatment. Many gingival lesions are associated with systemic disease and are, therefore, joint medical and dental problems. For example, gingivitis which exhibits tiny polyplike growths of granulation tissue protruding from the gingival crevices, especially if associated with an acetone breath, should cause the diagnostician to suspect diabetes. Dental treatments should be related to the progress that occurs under proper medical management.

In gingival lesions associated with mouth breathing, an active inflammatory reaction often is noted characterized by edema confined to the labial gingiva of the anterior teeth (Fig. 1). Extravascular cellular elements and fluids are expressed in this lush type of overgrowth. By contrast, the gingiva of the bicuspids and molars, which receives the stimulation of functional occlusion, is healthy. As a rule, there is an accumulation of some debris and calcareous deposits in the labial crevices; usually this is not sufficient to explain the extensive gingival involvement. Correction of the mouth breathing, perhaps by nasal operation, together with mouth hygiene and interdental massage, usually will result in cure.

In children's mouths there often is little more than a detachment of soft tissues from the crowns of the teeth accompanied by substantial overgrowth of tissue, which is at first lymphedematous in character and later fibrotic (Fig. 2). Such conditions develop slowly and are not the result of a single inflammatory reaction. The overgrowth is of a normal type of tissue and is induced when extravascular cells and fluids, ordinarily removed by the tissues, are left *in situ*. Capillary lymphatic drainage is sluggish; hence, after fibrosis develops, surgical excision is the best approach.

Often the pressure related to tipping stresses produced in abnormalities in centric closure of the mouth in Class II occlusion (Angle's classification) is an important factor (Fig. 3). Accumulated extravascular cellular elements and fluids are expressed in this abundant overgrowth. Reduction of the leverages, together with cleanliness while the swelling

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Fig. 1 • Edematous hyperplasia confined to labial gingivae of nonfunctional anterior teeth

is expressed in the form of lymphedema, is an effective procedure in preventing fibrosis. Cleanliness and massage, which are effective aids in forestalling the development of fibrotic hyperplasia, are necessary in the prevention of hyperplastic overgrowth of the gingiva when diphenylhydantoin sodium is administered (Fig. 4).

Other edematous swellings, also predominantly inflammatory, are lush, soft, fluid-containing overgrowths and may vary in color from deep red to pale blue. Pitting often is evident on compression. These overgrowths are painful and usually yield an exudate containing blood, serum or pus. The condition is associated with a neglected mouth and should be fully investigated. As a rule, from 15 to 20 varieties of bacteria inhabit the mouth at all times. The effect of such an environment too often obscures underlying systemic disturbances. The first line of approach is, therefore, cleanliness and massage unless a blood dyscrasia is suspected.

In hyperplasia associated with leukemia, the gingiva, although swollen and pale, may have the appearance of soft granulations (Fig. 5). The teeth may be loose as in scurvy, and the accumulated debris may produce a fetid odor. The mucous membranes of the mouth and throat may be red. Granulations extruding from adjoining tissues, such as in the nose, add to the suspicion. Often such conditions have been improperly diagnosed as Vincent's infection because of a superimposed erosive ulcer or two. Oxidizing agents obviously aggravate rather than improve such a condition. Hence, an analysis of the blood, including a differential count, is indicated before treatment is undertaken.

An analysis of the blood also should be made when purpura is present and there is spontaneous bleeding from the gingiva

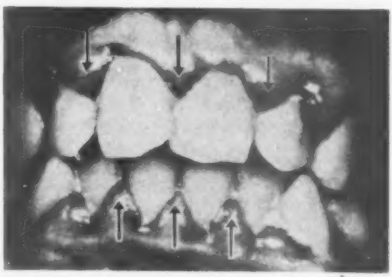

Fig. 3 • Edematous hyperplasia related to excessive tipping stresses produced in cases of abnormal paths of closure as the mandible moves from rest position to occlusion. Compare these functional teeth with nonfunctional anterior teeth in Figure 1

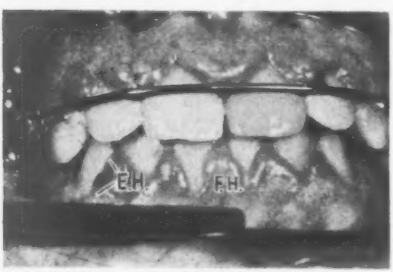

Fig. 2 • Hyperplastic overgrowth; transition from lymphedema to fibrosis. Lymphedema: edematous hyperplasia (E. H.); fibrosis: fibrosis hyperplasia (F. H.)

with little or no accompanying irritation (Fig. 6). In leukemia the white cell count is increased; in purpuric conditions the blood platelet count is low. These examples of blood dyscrasia are classical, but others associated with disarrangement of red blood cells, such as pernicious anemia, are more readily overlooked (Fig. 7).

It should be kept in mind that lush inflammatory swellings which involve the gingiva and are related to a vitamin C deficiency also occur in unclean mouths (Fig. 8). A dietary history and determination of vitamin C of the blood will establish whether this deficiency is a factor. The impregnation of the gingiva in dirty mouths with metallic salts of such metals as mercury and bismuth occurs because of a pre-existing lymphatic block. These are specific types of gingivitis which yield when there is a vitamin C deficiency to cleanliness and the addition of vitamin C to the diet and in the metallic impregnation cases to cleanliness plus elimination of the metallic salt.[1]

Systemic management may result in definite amelioration of marginal gingivitis, if treatment of dietary deficiency, metallic poisoning, blood dyscrasia, endocrine disturbances, and so forth, is included. In such instances, the lesion usually is diffuse in that the gingivae and the underlying connective tissues of many or all of the teeth are uniformly involved as occurs in gingivitis associated with pregnancy (Fig. 9). In general, such involvement is classed as "horizontal," whereas the isolated type with deep pockets is called "vertical." Such a classification is unfortunate unless used to designate the mode of progress for in both cases the irritating factor is mechanical, chemical, bacterial or physical in nature. This, together with the fact

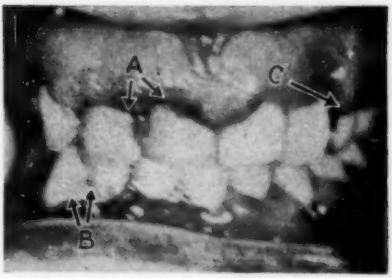

Fig. 5 • Myelogenic leukemia. A: Hyperplastic overgrowth, hemorrhagic phase; B: Erosive ulcer; C: Proliferating granulation tissue

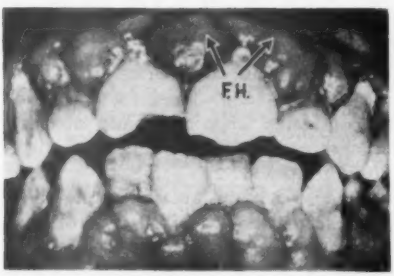

Fig. 4 • Hyperplastic overgrowth: complete fibrosis (diphenylhydantoin sodium). F. H., fibrosis hyperplasia

1. Lundquist, G. R. The zero crevice in the management of periodontal involvements. J.A.D.A. 27:837 (June) 1940.

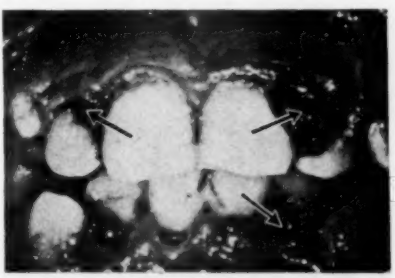

Fig. 6 • Purpuric gingivitis showing spontaneous hemorrhagic regions (courtesy of M. K. Hine, Indianapolis)

Fig. 7 • Pernicious anemia. Note calculus, dirt, debris and anemic appearance of the gingiva (courtesy of M. K. Hine, Indianapolis)

that the tooth is constantly erupting under function, probably accounts for the development of a gingival crevice or crevices in both these groups. If the irritation is long continued in a particular area in which there is excessive occlusal stress, the detachments will be deeper vertically here than elsewhere (Fig. 10). Hence, it may not be a difference of kind but possibly of time or degree or both.

These examples should be sufficient to show that the gingival lesion does not necessarily terminate when the medical aspect of the problem has been recognized and, when possible, corrected. The dentist is confronted then with a gingival crevice and later a periodontal pocket.

Patients with gingivitis caused by improper functional occlusion of the teeth are largely free from the abundant subgingival calculus deposits so prevalent in neglected mouths. The gingivitis often is expressed as a septal bulge, hemorrhagic on occasion, but usually it is not as lush and soft as that found in neglected mouths (Fig. 11). The more freely exudative character of these conditions in neglected mouths contributes to the formation of calculus. However, nature uses one pattern in reacting to irritation in the oral cavity; that is, classic inflammation, capillary leaks of cellular and fluid elements, plus dilatation and deformation of the capillaries with the accompanying lymphatic block.

Lack of functional use of teeth in a neglected mouth and overstress produce the same reaction. These factors should be considered when cause and effect relationships are being studied for diagnostic purposes.

Because mouths are habitually dirty, local accumulations of dirt, debris and bacteria are common to the lesions of dental caries and to periodontal involvements. Calculus deposits and trauma added to periodontal conditions are the differentiating factors between this condition and caries.

Given good occlusion both lesions probably would be largely prevented if every

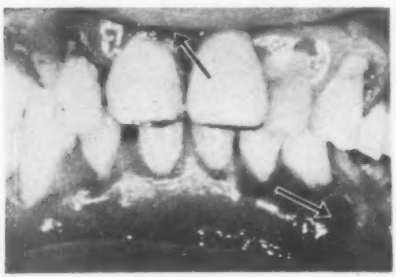

Fig. 8 • Gingivitis associated with vitamin C deficiency. Hemorrhagic regions are similar to those depicted in Figure 6

Fig. 9 • Gingivitis associated with pregnancy, showing lush semifluid, granulation tissue type of overgrowth

mouth were kept scrupulously clean and properly massaged. As a rule, no satisfactory attempt is made to eliminate dirt or debris and so a number of different types of disease are listed. Actually many variable conditions are produced by the presence of dirt, particularly when systemic disorders are present.

With dental caries, it is primarily the degradation of carbohydrate accumulation, which is acid producing, that seems to be at fault; while with periodontal involvements, products of partial protein cleavage are present which are alkaline in effect and toxic in character to the adjoining soft tissue.

In the former, the production of acid results in a decalcification of tooth structure. In the latter, the alkaline effect results in toxic irritation of the adjoining gingivae. This is characterized by capillary changes—that is, dilatation and deformation—and in extravascular infiltration of cellular elements and blood proteins, fluid in character (Fig. 12). Because of dirt and trauma, these inflammatory changes tend to persist, resulting in what might be called a "lymphatic block" (Menkin[2]). As a rule, nothing is done to check them even though they appear as early as the time of eruption of the teeth.

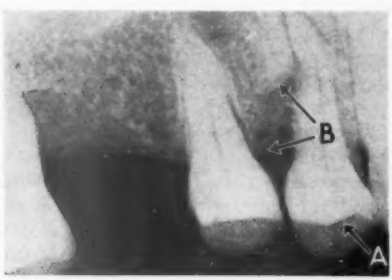

Fig. 10 • Vertical type of pocket formation related to excessive tipping stresses. Direction of force (A) with attendant resorption of bone, (B). Contact maintained mesially because supporting bone has not been resorbed as in B

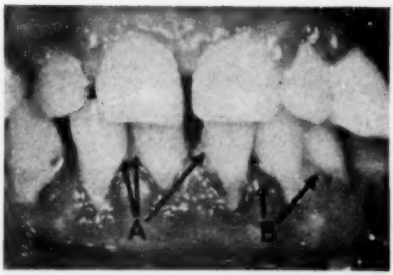

Fig. 11 • Chronic periodontitis, freely exudative type: A: Abundant calculus; B: Edematous hyperplasia

Tissue fluids and extravascular cells, which have become extravascular because the gingiva has been subject to repeated attacks of inflammation or long continued mild irritation, can be returned to blood vessels only via the lymphatics. To move such fluid through these vessels, an outside force often is necessary. Motion and massage are effective means of promoting lymphatic drainage.[3] Menkin[2] has demonstrated increased capillary leakage or permeability in inflammation and produced evidence that a dye injected into an area of sterile inflammation (aleuronat) remained in the site of injection and did not travel to regional lymphatics. He explained his findings on the basis of a local deposition of fibrin and thrombosis of lymphatics. Drinker and Yoffey[4] contend that capillary lymphatics in swollen tissue are widely distended because these vessels are attached to surrounding tissue by fine fibrils and, as swelling occurs, fluid passes freely into them and pulls them open. They point out that fibrin contracts, with the result

2. Menkin, Valy. Dynamics of inflammation; an inquiry into the mechanism of infectious processes. New York, Macmillan Co., 1940.

3. Lundquist, G. R. Periodontal involvement and its management. J.A.D.A. 32:298 (March) 1945.

4. Drinker, C. K., and Yoffey, J. M. Lymphatics, lymph and lymphoid tissue. Harvard University monograph in medicine and public health, no. 2. Cambridge, Mass., Harvard University Press, 1941.

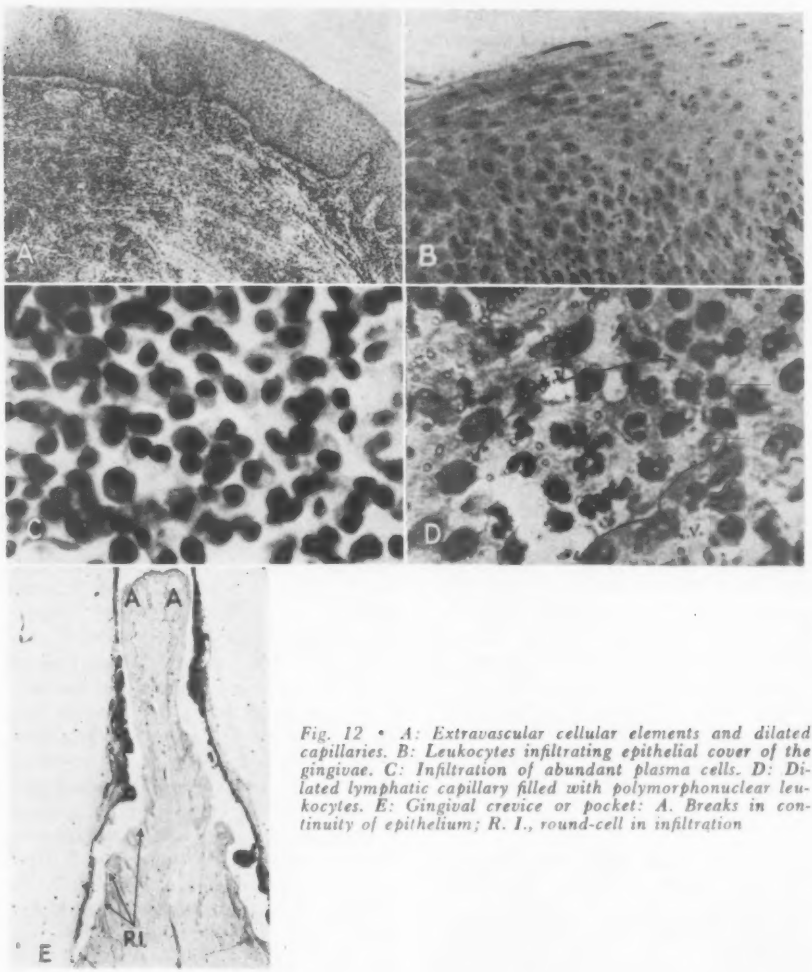

Fig. 12 • A: Extravascular cellular elements and dilated capillaries. B: Leukocytes infiltrating epithelial cover of the gingivae. C: Infiltration of abundant plasma cells. D: Dilated lymphatic capillary filled with polymorphonuclear leukocytes. E: Gingival crevice or pocket: A. Breaks in continuity of epithelium; R. I., round-cell in infiltration

that the formation of solid clots capable of blocking dilating vessels against a heavy head of lymph flow (fluid accumulation) is not easy, and may fail readily if inflammatory swelling is really great. They suggest that as inflammation progresses and tissue tension reaches an equilibrium, water and solutes of a small molecular size, but possibly of considerable toxicity, may be absorbed by the blood capillaries; whereas molecules of approximately the dimensions of serum globulin and those of larger size must either experience lysis, and so acquire small molecular dimensions, or must depend for their removal on lymphatic drainage or on phagocytosis with subsequent cellular migration.

Since capillary lymphatic drainage normally is sluggish it follows that the lymphatic capillaries are essentially the servant of the normal-moving human organism. Muscular activity is of prime importance in the movement of capillary lymph. It follows that without proper massage from the mastication of food or the interposition of such procedures as artificial massage to produce motion, the extravascular elements will continue to be left *in situ*. This factor helps to account for the chronic characteristics of periodontal conditions.

The large size of lymph capillaries and the lack of pressure difference between their contents and the tissue in which they are found make them open to influences of motion or massage and cause them to be pathways for the removal of foreign material.[3]

Lymph from normal skin owes its composition to transudation from capillaries in the skin, but lymph collected from mucous membranes also contains fluids, solutes and microscopic particles that have reached the lymphatics from the surface. The gingival crevice, with the series of breaks in continuity of the epithelial lining of the soft tissue sides of a periodontal pocket or gingival sulcus, makes this abundantly clear.

In children's mouths, there is little more than a detachment of soft tissue from the crown of the tooth accompanied by a substantial overgrowth of tissue, lymphedematous and later fibrous in character.[5]

In the adult, the detachment of soft tissues also includes separation of soft tissues from the root surface. The crevice is deeper. The toxic irritation of the adjoining soft tissues is more pronounced and more frequently repeated. Often the edematous fluid accumulation is less pronounced, and the extravascular cellular infiltration may be greater. Still later fibrous tissue is added. Treatment in both instances requires removal of the semisolid grayish white debris covering the gingival aspects of the crowns and roots of the teeth; but treatment requires more than this. Largely because mouths are habitually dirty, such accumulations constitute a source of long-continued toxic irritation, resulting in repeated attacks of inflammation. The lymphatic block accompanying this process also must be dealt with; hence, the local problem is twofold.

TREATMENT

The dirt—semisolid mucous particles—must be removed after each meal to prevent the recurrent irritation that produces the accompanying lymphatic block in the adjoining soft tissues. That this is an absolute necessity may be stated without qualification. Dentists and their patients have become so accustomed to the universality of dirt in the human mouth that the dentist makes and the patient accepts restorations in its presence.

This is unfortunate since prevention is seldom thought of before the consequence of such neglect is well advanced. The dentist seldom teaches his patient how to clean or massage his teeth and their investing tissues effectively. It follows that most toothbrushing is ineffective. It is done at the wrong time, and usually is improperly performed, so that today it has little value except for the fact that it has or produces a pleasant taste.

When a gingival crevice or sulcus develops or a detachment of the soft tissue from the tooth occurs there is a break in continuity of the epithelium accompanied by the clinical symptoms of inflammation. If inflammation is defined as a physiologic reaction to an irritant, it follows that the signs of inflammation will continue so long as the irritation persists. Consequently, if the irritant is dirt, in-

5. Lundquist, G. R. Periodontal conditions: etiologic factors in management. J.A.D.A. 34:663 (May 15) 1947.

fectious or chemical in nature, and is coupled with trauma, it is necessary to control and, when possible, eliminate such factors.

The effects of treatment may not be immediate since the inflammatory process may have been long continued. Under such conditions, the fluid and cellular elements that have leaked from the blood capillaries (increased capillary permeability) into the tissues are left *in situ*. If long neglected, these lush edematous swellings tend to become fibrous in character. Under such circumstances, their elimination often will require surgical management. Hence, it is much wiser to treat these swellings (lymphedema) before fibrous tissue is added to complicate the management of the problem.

As a rule the fluid and cellular elements are not removed from these edematous bulges for two reasons: first, the irritations which are physical, chemical or infectious in nature usually are repeated often and are long continued; and second, the fluid exudate contained therein tends to thicken as fibrin is added. The serum globulin particles thus developed are too large to pass through the lymphatic capillaries and therefore produce a stasis (lymphatic block).

The objective in treatment should be the removal of the extravascular fluid and cellular elements left *in situ*. Massage or pressure in the direction of lymph flow is one of the best artificial measures that may be used for moving extravascular fluid into lymphatics, thus moving lymph onward toward the blood stream. A combination of massage, passive movement and heat would be more effective still. Since gingival tissues are not amenable to judicious bandaging to produce pressure, as are arms or limbs or other parts of the body, zinc chloride packs are used. Massage is accomplished by toothbrushing and, more effectively, by interdental stimulation. Moist heat used in the form of hot normal salt solution for 15 minute periods is effective in acute inflammatory processes if rest periods of an hour are interposed between periods of use.

METHODS FOR USE OF ZINC CHLORIDE PACKS

All lower teeth are scaled and packed and the process is repeated on all upper teeth. A few strands of cotton are wound around a periodontal membrane explorer (Black) and dipped in an 8 per cent solution of zinc chloride. These packings then are interposed between the contact points and each buccal, labial and lingual bulge of the lower and upper teeth until the tissues blanch and are made anemic. After each pack is inserted, the mouth is irrigated with a normal saline solution. An 8 per cent solution of zinc chloride is a good astringent but is also a mild caustic. Irrigating as suggested will prevent irritation of adjoining tissues. If the labial, buccal or lingual crevices exist, with accompanying lymphedema, packings are worked directly into such crevices, blanching the adjoining free gingiva. This squeezes the cellular and fluid elements related to the inflammatory process out through the lymphatics, the normal drainage channels, toward the blood stream. The anemic effect lasts approximately 15 minutes. Thereafter the collateral circulation reestablishes fluid elements and the packings have no further value. Removal of the pack results in a resurgence of the circulation which, of course, is stimulating.

Ten days later, before the process is repeated, a thorough scaling is done. The patient is then taught how to use the interdental stimulator, which having been inserted interproximally, is directed toward the biting edge of the tooth as it passes through the septal space over the septal gingiva. The pressure in a lingual direction compresses and reduces the buccal bulge. From the lingual aspect, pressure is exerted buccally in like manner against the lingual bulge, blanching

and reducing it. The patient does the same thing each day in a modified way and achieves a result similar to that achieved by pressure activation of packs. Three weeks later the scaling and pack procedures are repeated.

As circulation resumes, the products of inflammation are absorbed. The development of deflecting contours as a consequence of the above procedure restores nature's ability to deflect food particles that pass the contact points through the labial, buccal and lingual embrasures, without injury to the septal gingiva. As a consequence, food accumulation, decomposition and toxic irritation of these tissues are prevented.

If these procedures are found to be ineffective three weeks later, then the question of functioning occlusion must be considered. If functional occlusion is faulty, it should be corrected where possible. The systemic factors which may be involved are reconsidered and the question of the surgical excision of the soft tissue side of the periodontal pocket also may be taken under consideration at this time.

Etiology and diagnosis of bacterial gingivitis including Vincent's disease

John R. Wilson, D.D.S., Columbus

The discovery of the presence of microorganisms in the oral cavity was made as early as the seventeenth century. Anton von Leeuwenhoek[1] studied microorganisms in deposits from his own teeth during the early development of microscopic procedures. Since that time the oral flora has been studied by many investigators, and modern methods of investigation have merely discovered more varieties.

Some of the organisms of the oral flora are regular inhabitants of the oral cavity, while others occasionally may be absent. Many things, such as food and water, are taken into the mouth and breathing sometimes is done through the mouth; therefore, transient bacteria may be in the mouth for only a short time. These transient bacteria are of no great significance. However, it is almost impossible to kill or remove all of the so-called resident bacteria. Some of these resident bacteria in the oral cavity are (1) cocci, (2) bacilli, (3) fusiform bacilli, (4) actinomyces, (5) vibrios, (6) spiral organisms, and (7) yeast organisms.[2]

Because there are some highly defensive mechanisms which make it difficult for the bacteria to enter the tissues, they usually are kept under control. It is when inflammation or injury interferes with these normal defenses that the bacteria may enter the tissues. Many authors have described distinctive bacteriologic infections in the mouth, such as streptococcal, staphylococcal, diplococcal and diphtheroid coccal. However, there has been

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1. Leeuwenhoek, A. Phil. Trans. Roy. Soc., London, May 20, 1684, no. 159, p. 568. Cited by Appleton, J. L. T., Jr., Bacterial infection, ed. 3. Philadelphia, Lea & Febiger, 1944, p. 301.

2. Coolidge, E. D., and Hine, M. K. Periodontia, ed. 1. Philadelphia, Lea & Febiger, 1951.

little significant research done which would help in making a satisfactory diagnosis of these distinct infections. Since it is still unknown which particular bacteria are responsible for Vincent's infection, it is almost impossible to differentiate these various infections. One of the reasons for not using the term "fusospirochetal infection" for the disease commonly known as Vincent's infection is because it has not been determined whether the fusiforms and spirochetes are solely responsible. For this reason, the term "acute necrotizing ulcerative gingivitis" has been used to describe a disease that has distinctive symptoms.

Necrotizing ulcerative gingivitis has been known for many years. There have been numerous references to this disease in dental literature, especially during wartime periods. Since it may develop into a gangrenous condition, it was originally thought to be a type of gangrene. Local, traumatic or systemic factors have made the tissue susceptible to Vincent's organisms. Usually a great number of spirochetal forms are found in a mouth infected by Vincent's organisms. Of these spirochetes, *Borrelia vincenti* seems to be the most important. Over a hundred types of these fusiforms have been cultivated. The fusiforms and spirochetes have been called the symbiotes of Vincent's infection, as well as vibrio, spirilla and anaerobic cocci, which have also been found in the lesions. Any situation which lowers the defenses or restricts the normal function of the oral tissues may allow these symbiotes to enter. These factors may be local, such as inflammation resulting from calculus, improper marginal fillings, poor contact areas, or lack of stimulation, or they may be systemic, such as any general systemic debilitated condition or dietary deficiency. The infection may begin on the tonsils and progress forward to the operculum of the last molar. Injury by drugs or excessive smoking or excessive use of alcohol also may be predisposing factors.

Acute necrotizing ulcerative gingivitis is characterized by a definite clinical picture. In its early stages, hyperemia and edema are in evidence. As the growth of bacteria progresses, the interdental papillae become necrotic and sloughing and ulceration result. The gingival margins also may be ulcerated. These ulcers are covered by a pseudomembrane which may be removed with an airblast.

One of the most important early diagnostic symptoms of acute necrotizing ulcerative gingivitis is pain on the slightest manipulation. Often a simple gingivitis is characterized by hyperemia and edema and rarely it will also be painful. This is important in making an early diagnosis, and this one diagnostic symptom should always be kept in mind since no other symptom may be present to differentiate between simple gingivitis and acute necrotizing ulcerative gingivitis. If this one symptom were noted more often, many cases of necrotizing ulcerative gingivitis might be checked before the other more pronounced symptoms developed. There may be an elevation of temperature to 103° F. or 104° F. and often lymphadenitis is present. General malaise and loss of appetite are common. There is a characteristic fetid odor of dead tissue.

Another predisposing factor which has not received proper emphasis is trauma. As is true in many other periodontal diseases, there may be more than one cause. In cases where it has been possible to remove infection except for one area of the mouth, examination will reveal that some cause of trauma existed in the area in question. This trauma may result from direct contact of the teeth and gingival tissue or from disharmony in occlusal forces involving one tooth with another.

Such areas as the third molar flap, periodontal pockets, or areas on restorations that cannot be cleaned, have been described by Box[2] as incubation zones and have been divided into two categories, primary and secondary zones. The primary zones are those in which the

bacteria multiply rapidly. The secondary zones, although less favorable for the growth of bacteria, become infected from the bacteria in the primary zones.

Since the clinical picture of the disease is so characteristic, it frequently is not necessary to resort to the bacteriological examination unless treatment does not bring a satisfactory response in a short time. Since the fusiforms and spirochetes occur in the mouth when Vincent's infection is not present, about the only value of the bacterial smear is to determine whether the organisms have become abnormally numerous.

CHRONIC VINCENT'S INFECTION

A sequel of untreated or unsuccessfully treated acute Vincent's infection frequently is found in mouths where there is an over-abundance of fusiforms and spirochetes. This is a chronic form of the disease which is usually associated with deep periodontal pockets.

The treatment for necrotizing ulcerative gingivitis is, of course, dictated by the etiology. The elimination of causative factors; for example, the removal of incubation zones and the correction of dietary deficiencies, is of more importance than medication by drugs. It is wise to use such drugs as penicillin or aureomycin[4] as an adjunct to treatment in order that the necrosis of tissue may be reduced to a minimum. However, most reoccurrences of this disease have been caused by failure to continue the débridement process and by incomplete elimination of the periodontal pockets and other incubation zones.[5] It should, therefore, be remembered that drugs are necessary, but only as adjuncts in the treatment process.[6,7]

COMMUNICABILITY OF THE DISEASE

The question of communicability has been discussed many times.[8] At the present time the pathogenicity of spirochete organisms has not been established by Koch's postulates. The criterion of the third postulate which states that pure cultures of the organisms, when injected in a suitable animal, must produce a disease, has not been fulfilled up to the present time. Pasteur stated that "there are some bacteria that cause a disease, but there are some diseases that bring about a condition that is ideal for the growth of some bacteria."[9] This statement is particularly significant in this particular instance. Schwartzman and Grossman[10] studied the communicability of Vincent's infection and reported that all attempts at direct transmission were unsuccessful. Others who have made these attempts have made similar reports. The Research Commission of the American Dental Association[11] appointed a special committee to consider the communicability of Vincent's infection. The report of the committee stated that Vincent's infection seemed to be one of a group of endogenous infections which, like subacute bacterial endocarditis or peritonitis caused by intestinal perforation, are induced by ordinarily harmless surface parasites under particular conditions. These diseases, as a group, are not communicable.

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10. Schwartzman, J., and Grossman, L. Vincent's ulceromembranous gingivostomatitis. Arch. Pediat. 58: 515 (Aug.) 1941.

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Herpetic gingivostomatitis

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Oral herpetic infections of man are relatively common and have been described under a variety of terms, such as "herpetic stomatitis," aphthous stomatitis," "ulcerative stomatitis," "primary herpetic gingivostomatitis," "recurrent herpetic stomatitis," and "recurrent habitual aphthae."

The use of such a large number of similar terms has caused a great deal of confusion in the diagnosis and treatment of the herpetic infections of the oral cavity. Many conditions not of herpetic origin have been placed in this disease group. Evidence of this confusion can be found in the writing of Dodd and co-workers[1] who, in 1938, reported a series of 88 cases of herpetic stomatitis in which they proved that the herpes simplex virus was the causative factor. In 1950, Dodd and Ruchman[2] published a paper entitled "Herpes Virus Not the Etiologic Agent in Recurrent Stomatitis." This article gives the impression that the cases were viral in origin but the laboratory findings reported did not verify the diagnosis. The casual reader, therefore, is left in a state of confusion. However, critical analysis of their reported cases is evidence of the confusion that exists generally since they described cases of allergic stomatitis and other types of stomatitis under the term "recurrent stomatitis."

Because of the existing confusion with regard to herpetic induced lesions, some attempt should be made to standardize the terminology and clarify the differential diagnosis.[3]

Primary (acute) herpetic gingivostomatitis is an acute primary infection produced by the herpes simplex virus as was shown by Dodd and co-workers[1] in 1938 when they isolated the virus from the saliva of children having the disease. Burnet and Williams[4] demonstrated specific herpetic neutralizing antibodies in the blood following this type of infection. Scott, Steigman and Convey[5] confirmed these findings and described the typical clinical characteristics of this infection. Numerous other workers have since isolated the virus from the lesions and observed the development of a high titer of neutralizing antibodies in the sera of convalescent patients.

The first entrance of herpes simplex virus into the body usually produces a gingivostomatitis, vulvovaginitis, or a varicelliform eruption. Once the virus has gained entrance into the body it probably persists throughout life,[6] even in the presence of antibodies in the blood which

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1. Dodd, K.; Johnston, L. M., and Buddingh, G. J. Herpetic stomatitis. J. Pediat. 12:95 (Jan.) 1938.

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3. Black, W. C. Etiology of acute infectious gingivostomatitis (Vincent's stomatitis). J. Pediat. 20:145 (Feb.) 1942.

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5. Scott, T. F.; Steigman, A. J., and Convey, J. H. Acute infectious gingivostomatitis. J.A.M.A. 117:999 (Sept. 20) 1941.

6. Burnet, F. M. Virus as organism; evolutionary and ecological aspects of some human virus diseases. Cambridge, Mass., Harvard University Press, 1945.

can quickly neutralize large quantities of virus *in vitro*. However, the virus is intracellular and is therefore protected from the specific antibodies in the blood, and can, if activated, produce recurrent disease. Cariell[7] has shown that the vesicles in recurrent herpes labialis contain considerable quantities of active herpes simplex virus in spite of a high titer of neutralizing antibodies in the blood. Recurrent attacks of herpetic stomatitis are not of the severe generalized type but are characterized by single or multiple discrete lesions and therefore present an entirely different clinical picture. Because of the difference in signs and symptoms between the initial infection and the secondary infection, the disease is divided into two phases—the primary infection which is termed "primary herpetic gingivostomatitis" and the secondary infection termed "recurrent herpetic stomatitis." These two phases of the infection will be discussed separately.

PRIMARY HERPETIC GINGIVOSTOMATITIS

This is an acute infectious disease caused by the herpes simplex virus. The first study of the herpes simplex virus was in 1912 when Grüter[8] inoculated the scarified cornea of a rabbit's eye with material from the vesicular lesions of a patient suffering from keratitis. Typical purulent keratoconjunctivitis was produced and the disease was transmitted from one animal to another. In the eye material, the characteristic Lipschütz inclusion bodies were demonstrated. Dodd, Buddingh and Johnston,[1] in 1938, isolated the virus from patients exhibiting the typical acute symptoms of herpetic stomatitis. Burnet and Williams[4] stated that following this type of infection, specific herpetic neutralizing antibodies appear in the blood. Scott, Steigman and Convey[5] isolated the virus by corneal inoculation in 17 of 21 cases tested. The blood of 19 of the 21 cases developed neutralizing antibodies during the convalescent stage of the disease. Boswell[9] has studied the virus with the electron microscope.

Communicability of the disease has been well established by observation; for example, Scott, Steigman and Convey[5] reported a history of contact in almost 50 per cent of their cases. Familial epidemics, of which there were three reported in their series, is evidence of communicability. Similar findings are reported by other authors after observing a large series of cases. The fact that at least three quarters of the adult population has been infected at some time by the herpes simplex virus is indicative of its communicable nature.[9, 10, 6]

INCIDENCE

In about 70 per cent of the population the initial infection is during infancy and early childhood.[4] The highest incidence of the disease is between the ages of 1 and 3.[5] It is infrequent in young people in the late teens and uncommon in adults. In about 1 per cent of all infections, the initial attack produces severe disease.[4] Evidence of differences of incidence among sex or racial groups is lacking. The almost universal presence of herpes virus neutralizing antibodies in the serum of adults indicates the incidence of this infection.

CLINICAL SIGNS AND SYMPTOMS

Primary herpetic gingivostomatitis is an acute infection of a severe and generalized nature. The initial infection frequently is preceded by some disease

7. Blank, H. Virus diseases affecting the skin. Acta Dermat.-venereol. 29:75, 1949.

8. Grüter, W. Experimentelle und klinische Untersuchungen über den sogenannte Herpes Corneae. Klinische Monatsblätter f. Augenheilkunde 65:398, 1920.

9. Boswell, F. W. Electron microscope studies of virus elementary bodies. Brit. J. Exper. Path. 28:253 (Aug.) 1947.

10. Braitman, M. Recurrent aphthous stomatitis treated with aureomycin. J. M. Soc. New Jersey 47:160 (April) 1950.

process such as an upper respiratory infection, infectious mononucleosis, or a gastrointestinal upset, which lowers the customary resistance of the individual to the virus. The onset frequently is insidious and the early symptoms are mild and nonspecific, hence they are not given consideration.

The infant cries without apparent reason, is irritable, fretful, drools excessively, and frequently refuses food and fluids. Older children are irritable, fretful, quarrelsome, lack normal interest in play, are lethargic and complain of headache, soreness in the neck, and pain on swallowing. Within a day or two the articulate patients complain of a sore mouth following the appearance of the ulcers. The characteristic physical findings during the prodromal or pre-exanthem stage of the disease are swelling and tenderness of the submental, submaxillary, and superficial cervical lymph nodes. The patient often complains of soreness of the neck. Sore throat is a frequent early complaint because of the involvement of the lymphoid tissue of the pharynx, especially the tonsil. There is an increase in temperature ranging from 99°F. to 105°F. with greatest elevation in the younger patients. The temperature remains elevated from 3 to 5 days and usually subsides rapidly following the appearance of the mucous membrane or skin eruptions. In some patients, especially the extremely young, the symptoms are complicated by dehydration and acidosis. After the prodromal stage, the typical oral manifestations occur, causing the patient to seek aid. The gingival tissues are swollen, intensely red, painful, and show considerable tendency to bleed. This frequently leads to confusion with Vincent's fusospirochetal infection. The tongue, buccal mucosa, palate, and pharynx are all involved to a variable degree depending on the severity of the disease. Vesicles and whitish spots of various size appear. The thin walled vesicle and the delicate membrane rapidly degenerate and a discrete shallow ulcer develops, the border of which is undermined and ragged. The conversion of the vesicle or plaque to an ulcer takes place so rapidly that the vesicles are only occasionally observed. The base of the ulcer, at first clean, soon is covered by a yellowish gray pseudomembrane. The inflammatory reaction at the border of the ulcer is severe and sharply circumscribed. The red ring about the ulcer frequently is referred to as the "inflammatory halo." The ulcers rapidly become intensely painful. When several vesicles appear in a small area they coalesce to form a large irregular ulcer. The ulcers occasionally are found on the gingiva and the pharyngeal mucosa. In extremely severe cases, lesions may appear in the nasal mucosa, the larynx and the esophagus. Fetor oris always is present during the course of the disease.

The general feeling of the patient usually is improved and the temperature drops after the appearance of the ulcers. The soreness of the mouth and throat decreases in about three days when the ulcers begin to show evidence of healing. The entire mouth usually is normal in 7 to 14 days.

The disease varies in severity, being most severe in infants whose temperature ranges from 105°F. to 106°F. It is accompanied by acidosis and dehydration. In a few instances the patients develop meningoencephalitis.[11,12] Death results in a few cases.

PATHOLOGY

The changes in the buccal mucosa, skin, cornea and central nervous system are essentially the same, and indicate that

11. Smith, M. G.; Lennette, E. H., and Reames, H. R. The isolation of the virus of herpes simplex and the demonstration of intranuclear inclusions in a case of acute encephalitis. Am. J. Path. 17:55 (Jan.) 1941.

12. Zarafonetis, C. J. D., and others. Fatal herpes simplex encephalitis in man. Am. J. Path. 20:429 (May) 1944.

the virus is ectodermotropic.[13] The first change in the mucosa in herpetic gingivostomatitis is a ballooning degeneration of the epidermal cells and secondary inflammatory infiltrations in the submucosa. In the areas of degeneration the epithelial cells contain characteristic inclusion bodies. The Lipschütz inclusion bodies are within the nucleus and have a homogeneous ground glass texture, stain red or lavender, and cause the nucleolus and chromatin to collect at the margin of the nucleus. In some regions there is edema and hydropic degeneration with accumulation of fluid which produces typical vesicles filled with clear fluid containing the herpes simplex virus in large quantities. Polymorphonuclear cells appear in the submucosa and the vesicular fluid. The vesicles in the mucosa are thin walled and rupture almost as soon as they appear leaving a shallow ulcer with an irregular undermined border. The ulcer becomes secondarily infected and painful. An abundant fibrous exudate with entrapped inflammatory cells and cell debris covers the ulcer base producing the characteristic clinical yellow color. There is slight simple necrosis of the submucosa and heavy infiltrations of leukocytes. In 48 to 72 hours there is evidence of epithelial regeneration and the lesion heals rapidly without scarring.

LABORATORY FINDINGS

Smears of the oral cavity show no change in the normal oral flora but if the smear material is placed on the scarified cornea of a rabbit eye a characteristic purulent keratoconjunctivitis develops within 48 to 72 hours. The corneal epithelium shows the typical ballooning degeneration and the presence of nuclear inclusions.[14] Successive inoculations produce the characteristic corneal changes and a typical encephalitis with inclusion bodies in the brain.

During the convalescent period specific herpetic neutralizing antibodies develop in the blood. The antibodies remain in the blood permanently. By means of the embryonated hen's egg the virus may be directly isolated in material which has been taken from patients' mouths.[15] In some cases this replaces the rabbit cornea. Material from the mouth is mixed with penicillin and streptomycin and inoculated into the chorio-allantoic membrane. With proper technic each infectious unit induces a proliferation of the epidermal cells with the production of a clearly discernible plaque.[13] An important use of the egg has developed from Nagler's[16] demonstration that 72 hours after inoculation of the yolk sac with the herpes virus, a high titer of the virus appears in the amniotic fluid. This clear fluid, almost free from normal egg proteins, then can be used as a skin test antigen. Forty-eight hours after interdermal injection a tuberculin-like reaction appears in the skin of patients who have been infected with herpes simplex virus. This skin test is comparable to the Frei test and parallels the antibodies in the blood. It is positive for the majority of human adults.[16] Cross-immunity between smallpox virus and herpes simplex has not been demonstrated and it has been shown that the presence of vaccinia virus in a cell does not prevent the entrance of the herpes simplex virus. Both viri may infect the same cell simultaneously.[17, 4] Significance of this evidence of the absence of cross-protection will be discussed under treatment.

13. Anderson, K. Pathogenesis of herpes simplex virus infection in chick embryos. Am. J. Path. 16:137 (March) 1940.

14. Lipschütz, B. Die Einschlusskrankheiten der Haut. (Das filtrierbare Virus in der Dermatotologie). In Handbuch der Haut und Geschlechtskrankheiten, vol. 2. Jadassohn, J., editor. Berlin, Springer, 1932.

15. Rose, H. M.; Pearce, E., and Malloy, E. Effect of penicillin and streptomycin on bacterial contamination of chick embryos inoculated with unfiltered sputums. Proc. Soc. Exper. Biol. & Med. 62:124 (June) 1946.

16. Nagler, F. P. O. A herpes skin test reagent from amniotic fluid. Australian J. Exper. Biol. & M. Sci. 24:103 (June) 1946.

17. Anderson, K. Dual virus infection of single cells. Am. J. Path. 18:577 (July) 1942.

DIFFERENTIAL DIAGNOSIS

Primary herpetic gingivostomatitis must be differentiated from Vincent's infection, exanthemas of acute infectious diseases such as chickenpox and measles, and chemical and allergic stomatitis.

The oral tissues may be predisposed to Vincent's infection by systemic disease. The disease is accompanied by fever and lymphadenopathy, is characterized by soreness, swelling and bleeding of the gums, and therefore simulates a primary herpetic gingivostomatitis. The fever and lymphadenopathy associated with Vincent's infection usually does not precede the soreness of the oral cavity as in herpetic gingivostomatitis. Necrosis of interdental tissues always is present in Vincent's infection but never is found in herpetic gingivostomatitis. The absence of interdental necrosis is the most important point of differential diagnosis.

The oral lesions of the exanthemas usually are confined to the mucosa and the gingiva is not involved. The simultaneous or alternate appearance of skin lesions is important in differential diagnosis. In Kaposi's varicelliform eruption, which is an initial infection of the skin with the herpes simplex virus, there may be associated oral lesions.

Chemical stomatitis may be vesicular and ulcerative but does not have the associated elevation of temperature and lymphadenopathy.

Allergic stomatitis may resemble closely the oral manifestations of primary herpetic gingivostomatitis but the associated constitutional symptoms are absent.

TREATMENT

Primary herpetic gingivostomatitis, because it is a communicable infection of a self-limited type, does not respond to therapeutic measures of abortive or curative character. The disease has certain prodromal, active, and convalescent phases which are unalterable. Since drugs are not yet available which are effective in this disease, the treatment must be supportive and symptomatic. Nonirritating bactericidal drugs such as nitromersol, acriflavine or mercocresols may be used to reduce secondary infection. Penicillin may be used for the same purpose. Aureomycin, in adequate doses, administered at the onset of the disease, may alter slightly the course of the disease but is of greatest value in reducing secondary infection. Antipyretics should be used to reduce the temperature and forced fluids should be used to prevent dehydration. In extremely young patients dehydration presents a major problem and is best treated in a hospital. Patients should be isolated because of the communicable character of the disease.

SECONDARY HERPETIC STOMATITIS

This condition is termed in the literature "chronic recurrent herpes," "recurrent herpetic stomatitis," "habitual aphthae" and "chronic aphthous stomatitis." There are numerous other designations as well.

The incidence of secondary herpetic infections is extremely high. The studies of Burnet and Lush[18] on the presence of herpes neutralizing antibodies in adult blood serum indicate that infection by the herpes virus is almost universal, and verify clinical observations regarding the high incidence of herpetic infection. It is unusual to find a middle-aged adult who has not had at least one cold sore or canker sore. In a great many individuals there is a sporadic recurrence of these lesions, in some instances at frequent intervals.

ETIOLOGY

The cause of recurrent herpetic lesions is the herpes simplex virus as has been

18. Burnet, F. M., and Lush, D. Herpes simplex: studies on the antibody content of human sera. Lancet 1:629 (March 18) 1939.

demonstrated by corneal inoculations in rabbits and studies on embryonated hens' eggs. The virus is intracellular, ectodermotropic in character, and is present in the cell indefinitely following the initial infection in spite of the presence of neutralizing antibodies in the blood. The residual virus may be activated at intervals and produce discrete lesions in a specific area of the body. Evidence indicates that many factors are involved in the activation of the virus. Herpetic lesions frequently appear following dental appointments because of the minor trauma produced by manipulation of the tissue. Mild exposure to sunlight is responsible in many instances. The frequency with which the lesions have been associated with upper respiratory infection is indicated by their being called "cold sores." The elevation of body temperature, systemic diseases, of which infectious mononucleosis is an example, allergic reactions, and menstruation are systemic factors which activate the virus.

CLINICAL FINDINGS

The appearance of herpetic lesions is preceded by a localized burning sensation accompanied by a slight swelling and in a matter of hours a small vesicle or group of tiny vesicles appears. On the mucous membrane surfaces the vesicles rupture early leaving a shallow area with an irregular undermined border. The ulcer becomes secondarily infected and painful. On the mucous membrane surface the vesicle is of such a transient nature that the extremely painful ulcer is often the first clinical symptom. Soon after the ulcer appears it is covered with fibrin which produces a yellowish membranous appearance to the base of the ulcer. Following secondary infection an inflammatory red halo develops at the border of the ulcer. The ulcer is tender for a few days and heals spontaneously in 7 to 10 days without scarring. On the skin surface the vesicles have a thicker membrane and are more resistant to rupture. Within 24 hours they either rupture and crust or become desiccated and crusted. At this time they usually become asymptomatic and heal in 7 to 14 days without scarring. Special names have been given the lesions to indicate the anatomic location. Those occurring on the lip are called "herpes labialis" and those on the mucous membrane "aphthae." When they occur at the mucobuccal fold they are termed "aphthae. fissuratum." Sialorrhea and regional lymphadenopathy may accompany the lesions during the period of active ulceration.

LABORATORY FINDINGS

Neutralizing antibodies always are found in the blood serum because of previous infection. The changes seen in the tissue are the same as those found in the lesions associated with the primary phase of the disease.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis of this phase of the disease is difficult and this is responsible for much of the confusion existing in the literature. The lesions of periadenitis mucosa necrotica recurrens are similar to the herpetic aphthous ulcers and are recurrent. This makes differential diagnosis difficult. Many persons have reported that chronic recurrent aphthous stomatitis is nonviral in origin. This is because of errors in differential diagnosis. The lesions of periadenitis mucosa necrotica recurrens occur periodically on gland-bearing mucosal surfaces as a slight plaque with swelling and a slight burning sensation as the initial symptom. The small yellowish plaque is in the area covering a small mucous gland and is produced by necrosis of the mucosa which rapidly sloughs out leaving a somewhat craterlike ulcer. The area is covered with fibrin and becomes second-

arily infected causing it to be painful and to develop an intensely inflamed periphery similar to the inflammatory halo about the typical herpetic lesion. The ulcer of periadenitis mucosa necrotica recurrens is deeper but in all other respects is clinically identical with the herpetic lesion. The ulcers of periadenitis mucosa necrotica recurrens may be larger, in some instances being a centimeter in diameter. In periadenitis mucosa necrotica recurrens the larger and deeper ulcers heal slowly and produce scar tissue. Examination of the mouths of such patients reveals scarring in the area of the involved mucosa. The longer healing period and the evidence of scarring is of great importance in arriving at a differential diagnosis.

TREATMENT

Two measures for the control of recurrent herpetic stomatitis have been utilized—the relief of symptoms being one, the prevention of lesions the other.

Local therapy consists of alleviating pain and promoting healing. Topical application of caustic drugs such as silver nitrate and phenol has been used extensively. The topical application of antiseptic drugs and antibiotics, especially aureomycin, has been used to reduce secondary infection and promote healing. Local treatment is entirely palliative.

Several different methods have been tried to prevent recurrence of the lesions. In cases of herpes menstrualis attempts to prevent activation of the virus by the administration of diethylstilbestrol prior to the onset of the menses has met with some success. Large doses of vitamin B[19] complex have been suggested in the belief that the virus was neurotrophic and it was thought that the presence of vitamin B would prevent activation. The use of smallpox vaccine[20] has been suggested by many on the theory that a cross immunity between the herpes and vaccina virus existed and the presence of vaccina virus in the cells would prevent the entrance of the herpes virus into the cells, a mechanism demonstrated in certain other virus diseases. Animals with strong immunity to one virus, however, are susceptible to infection with the other[21] and the two viruses may infect the same cell simultaneously.[17, 22] This negative evidence from the laboratory probably explains the inconsistent results obtained from this form of therapy.

Aureomycin has been used prophylactically both as an ointment and by oral administration. As in other forms of therapy the results are not uniformly good. Blank and Brody[23] reported a study of 11 cases of recurrent herpetic stomatitis treated by psychotherapy in which excellent results were obtained in all but one case. The lack of uniformly good results with numerous forms of therapy indicates the need for better differential diagnosis and for further controlled investigation of etiology and treatment.

CONCLUSION

Primary herpetic gingivostomatitis and recurrent herpetic stomatitis represent the initial and recurrent form of herpes simplex virus infection, or may be considered as the acute and chronic phases of this disease. There is need for further clinical and laboratory study of these two conditions.

The clinical recognition of the acute phase of the disease is important because of the frequency with which it is confused with Vincent's infection.

19. Burket, L. W., and Hickman, G. C. Oral herpes (simplex) manifestations: treatment with vitamin B complex. J.A.D.A. 29:411 (March) 1942.

20. Woodburne, A. R. Herpetic stomatitis. Arch. Dermat. & Syph. 43:543 (March) 1941.

21. Boak, R. A.; Carpenter, C. M., and Warren, S. L. Symptomatic herpetic manifestations following artificially induced fever. (Abst.) J. Bact. 27:83. 1934.

22. Syverton, J. T., and Berry, G. P. Multiple virus infection of single host cells. J. Exper. Med. 86:145 (Aug.) 1947.

23. Blank, H., and Brody, M. W. Recurrent herpes simplex: psychiatric and laboratory study. Psychosom. Med. 12:254 (July-Aug.) 1950.

Fibrous hyperplasia of gingiva

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Normally, gingival tissues almost entirely fill the interproximal spaces between each tooth, beginning at the contact area between the teeth and surrounding each tooth with a thin delicate free gingiva which continues apically and laterally in a smooth curve. Frequently, however, instances will be found where there is an increase in the size of the gingiva, with soft tissue overfilling the interproximal spaces and ballooning out beyond the teeth. The enlarged gingiva may be localized to one papilla, or may involve several or all the gingiva throughout the mouth. The enlargement does not involve the vestibular mucosa (Fig. 1, above left). Glickman[1] has grouped all gingival enlargements into six classes: (1) inflammatory gingival enlargements; (2) noninflammatory hyperplastic gingival enlargements; (3) combined gingival enlargements; (4) conditioned gingival enlargements (hormonal, leukemic or associated with vitamin C deficiency); (5) neoplasms, and (6) developmental gingival enlargements. This classification is quite complete, but from a clinical point of view cases of gingival enlargement can be considered of two general types of tissue and may be termed "inflammatory hyperplastic gingivitis" and "fibrous hyperplasia of the gingiva."

In inflammatory hyperplastic gingivitis, the enlarged gingiva is soft, edematous, hyperemic or cyanotic, sensitive to touch, not stippled and can be made to bleed quite easily (Fig. 1, above right). In fibrous hyperplasia of the gingiva, the enlarged tissue is firm, dense, resilient, normal in color or slightly paler than normal, insensitive, and not easily traumatized (Fig. 1, below). Frequently there will be a combination of the two types of enlargement.

Both types of gingival enlargements apparently can result from chronic local irritations. In addition, the inflammatory type may be associated with vitamin C deficiency (Fig. 1, above right), leukemia, a hormonal unbalance, or pregnancy, and the fibrous type may be "idiopathic" or associated with the use of the drug diphenyhlydantoin sodium. This paper is limited to a consideration of the fibrous types of gingival enlargements.

TERMINOLOGY

Increase in the bulk of gingiva usually is labeled "hypertrophy" since common usage of the word has made it mean "increase in size." However, hypertrophy is correctly defined as "increase in size of a structure due to an increase in the size of the individual cells involved," so the preferred term for the condition under consideration is "hyperplasia," which is defined as an increase in bulk of tissue due to an increase in the number of cel-

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1. Glickman, I. Basic classification of gingival enlargement. J. Periodont. 21:131 (July) 1950.

lular elements. Fibrous hyperplasia of the gingiva may vary in degree from a slight increase in gingival bulk to definitely disfiguring enlargements, and may be grouped into three classes: (1) small enlargements associated with some local irritations on tissues predisposed to fibrous proliferation, (2) idiopathic fibrous hyperplasia, so-called "fibromatosis," and (3) those caused by the drug diphenylhydantoin sodium. Following is a discussion of these three types, with some examples of each.

FIBROUS HYPERPLASIA OF GINGIVA CAUSED BY LOCAL IRRITATION

Clinical Description • Mild cases of fibrous hyperplasia of the gingiva are seen frequently. Clinical examination will reveal this hyperplasia as an increased bulk of gingiva in the interproximal papillae or as a prominent festoon. It may be located in one area, in one quadrant, in the maxilla or the mandible, or it may be general (Fig. 2).

The tissue is a cool coral pink, similar to normal gingiva, or slightly paler. On palpation the enlargement will be found to be firm, unyielding, painless, and with no tendency to bleed unless, because of malhygiene or local irritation, some incidental inflammation also is present (Fig. 2, C and D, and Fig. 3).

The patient seldom will complain about the fibrous hyperplasia in its mild form. Such areas do not continue to expand if local irritations are removed or reduced.

Etiology • This mild form of hyperplasia frequently occurs in gingiva which is being subjected to some form of local irritation such as results from malocclu-

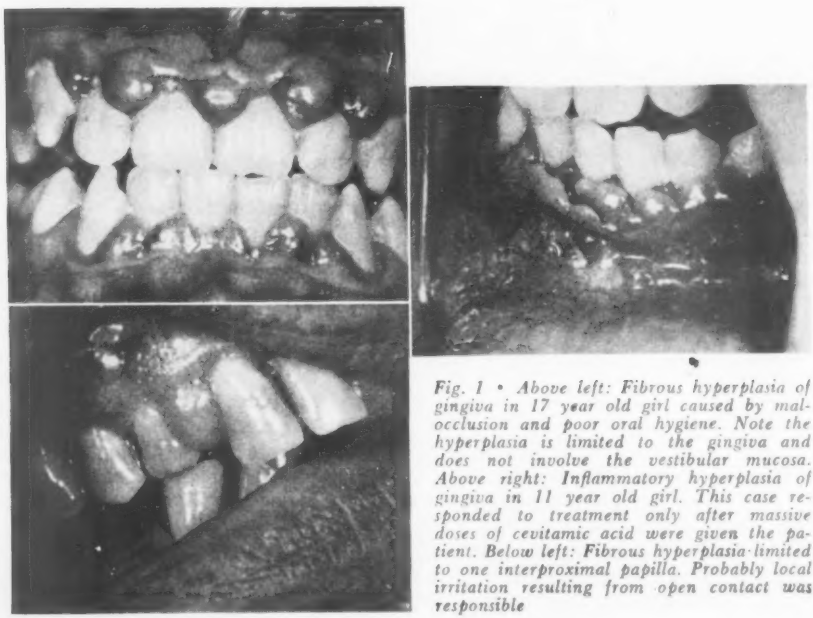


Fig. 1 • Above left: Fibrous hyperplasia of gingiva in 17 year old girl caused by malocclusion and poor oral hygiene. Note the hyperplasia is limited to the gingiva and does not involve the vestibular mucosa. Above right: Inflammatory hyperplasia of gingiva in 11 year old girl. This case responded to treatment only after massive doses of cevitamic acid were given the patient. Below left: Fibrous hyperplasia limited to one interproximal papilla. Probably local irritation resulting from open contact was responsible

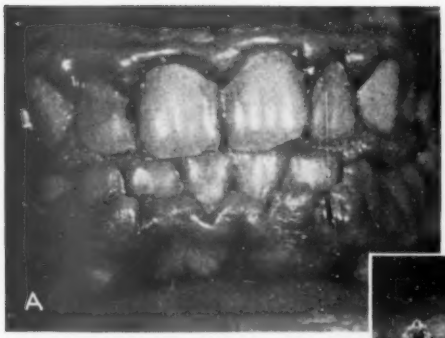
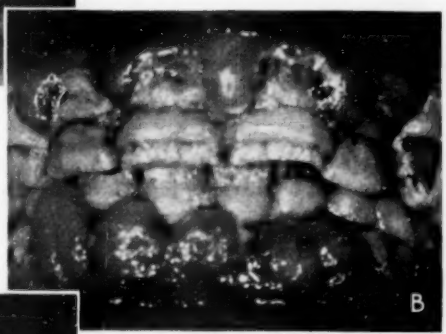


Fig. 2 • A: Generalized fibrous hyperplasia with some local inflammatory changes present. The etiology was unknown



B: Fibrous hyperplasia of gingiva in young adult, probably caused by local irritation. Acute necrotizing gingivitis was also present when this picture was taken

C: Chronic fibrous hyperplasia in young adult

D: Showing same patient after improvement of oral hygiene and removal of excess fibrous tissue overlying upper central incisors

sion, mouth breathing, poor contact areas, or irritation from ill-fitting crowns, clasps, prosthetic or orthodontic appliances or from poor oral hygiene. In such cases it is logical to assume that local irritations are the cause, but the fact

that in many other instances just as much local irritation exists without the hyperplasia suggests the presence of some predisposing factor which results in proliferation of fibrous tissue rather than a simple inflammatory reaction. Why one case shows proliferation of fibrous tissue, and another does not, depends on factors which are as yet unknown.

Marshall-Day and Shourie[2] reported that they found what they labeled "proliferative" hyperplastic gingivitis in from 77 per cent to 99 per cent of the Indian children studied. They stated the etiology to be "mixed" with a background of systemic predisposing factors such as metabolic or endocrine disturbances, or mineral or vitamin deficiencies. They found no evidence that oral hygiene played a major role in the etiology of hyperplastic gingivitis. Anderson[3] also reported a high incidence of fibrous hyperplasia among the Chinese.

No one has reported as high an incidence of fibrous hyperplasia in this country, but instances can be seen almost every day in a large dental clinic.

Treatment • Removal of all local irritations and institution of good oral hygiene is indicated, but if a large bulk of fibrous tissue is present, the tissue should be removed surgically. The treatment of the dense tissue by electrocoagulation or chemical cautery is difficult; gingivectomy is the treatment of choice (Fig. 4, above).

Prognosis • There is some tendency for such overgrowths of gingiva to recur (Fig. 4, below) although maintenance of excellent oral hygiene may prevent, postpone or minimize recurrences of the enlargement.

2. Marshall-Day, C. D., and Shourie, K. L. Hypertrophic gingivitis in Indian children and adults. Ind. J. M. Research 35:261 (Oct.) 1947.

3. Anderson, B. G. Hypertrophic gingivitis among Chinese. Nat. M. J. of China 15:453 (June) 1939.

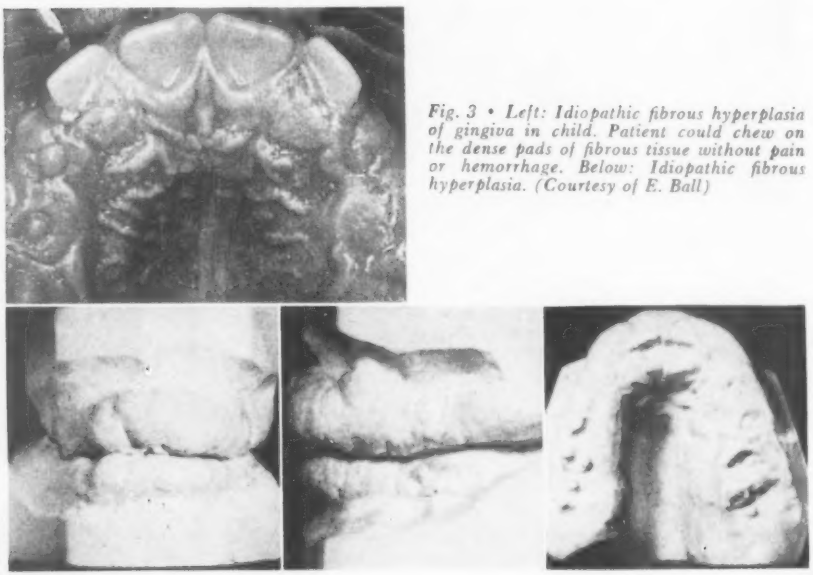


Fig. 3 • Left: Idiopathic fibrous hyperplasia of gingiva in child. Patient could chew on the dense pads of fibrous tissue without pain or hemorrhage. Below: Idiopathic fibrous hyperplasia. (Courtesy of E. Ball)

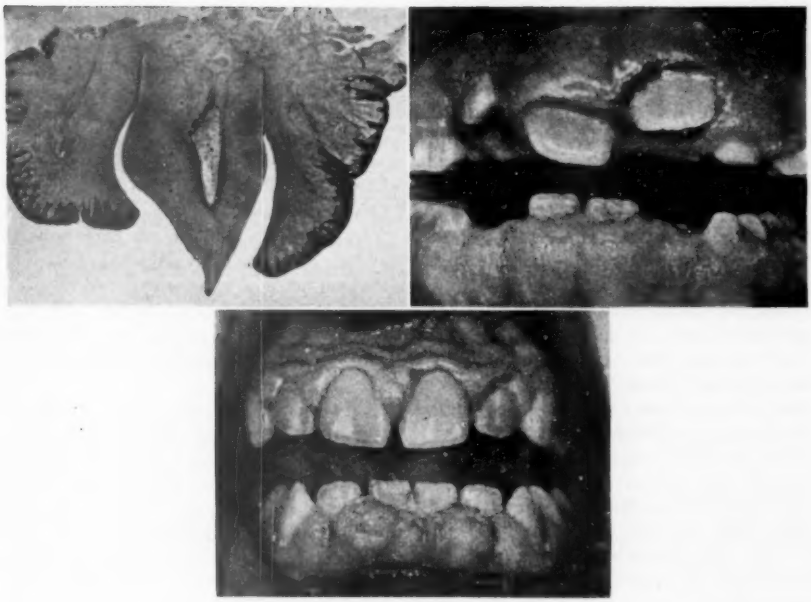


Fig. 4 • Above left: Deciduous upper cuspid with attached fibrous hyperplastic tissue. (Courtesy of B. Orban.) Above right: Crowns of permanent teeth exposed to allow them to erupt through the dense fibrous tissue. Below: Fibrous hyperplasia caused by diphenylhydantoin sodium. Note typical stippled or pebbled surface. (Rowe Smith's case; courtesy of B. Orban)

IODIOPATHIC FIBROUS HYPERPLASIA OF THE GINGIVA

Patients are seen occasionally with gingival tissues so enlarged that the teeth are covered, or if the enlargement is present before eruption of the teeth, the dense fibrous tissues may interfere with eruption. Many such cases have been described and termed "primary hypertrophy,"[4] "fibromatosis gingivae,"[5] "elephantiasis,"[6] "idiopathic fibromatosis,"[7] "congenital macrogingivae,"[8] "gingivoma,"[9] or "hereditary hypertrophy of gums."[10] Fibromatosis is not a good term sinc "osis" means degeneration and this is not a degenerative condition. Idiopathic fibrous hyperplasia of gingiva seems better.

Cases have been reported in the literature by Mirolli,[10] Ruggles,[4] and Blayney,[11] as well as by many others.

Clinical Description • In a typical case of idiopathic fibrous hyperplasia, the most striking observation is the large mass of firm, dense, resilient fibrous tissue that

4. Ruggles, S. D. Primary hypertrophy of the gums. J.A.D.A. 11:1204 (Dec.) 1924.

5. Bernier, J. L. Manual for the differential diagnosis of oral lesions. St. Louis, C. V. Mosby Co., 1942.

6. Prinz, H., and Greenbaum, S. Diseases of the mouth and their treatment. Philadelphia, Lea & Febiger, 1939, p. 163.

7. Ziskin, D. E., and Silvers, H. F. Two cases of subgingival proliferative lesions of probable hormonal origin. J. Periodont. 14:89 (July) 1943.

8. Byars, L. F., and Sarnat, B. G. Congenital macrogingivae (fibromatous gingivae) and hypertricosis. Am. J. Orthodont. & Oral Surg. 31:48 (Jan.) 1945.

9. Ball, E. L. Gingivoma, or elephantiasis of the gingivae. Bul. Cincinnati D. Soc. 11:15 (June) 1941

10. Mirolli, A. La elefantiasi familiare delle gengive. Arch. Ital. di Chir. 29:401, 1931.

covers the alveolar ridges (See Fig. 3). In the most severe cases, both maxillary and mandibular ridges are covered with tissue so dense that on palpation one suspects some bony overgrowths, although roentgenographic studies of the alveolar bone show it to be essentially normal in quantity and quality. The hyperplastic tissue is essentially normal in color, is insensitive, and does not bleed easily. The patient complains only of the deformity.

In one case reported by Ruggles,[4] the gingivae were so large the lips were averted, and the central portion of the face pushed forward. The fibrous mat of tissue was almost one inch thick in the molar area, and three-quarters of an inch thick in the anterior region, and the arches were about one inch deep. The case reported by Ball was almost as severe (Fig. 3, below).

The hyperplasia may be noted at an early age; Blayney[11] reported some cases were noted at birth; Perkoff[12] described a case in a child of 3; Heath[13] presented a case in a child of 4½ years of age which began when the patient was 2½ years old. In most of the severe cases, the teeth do not erupt normally through the dense fibrous tissue, or, when they do erupt, may be almost covered by the tissue.

Etiology • The cause of the overgrowth of gingival tissue is not known, although it is probably genetic.[8] Murray[14] reported on one family in which there were three persons with it, the youngest of the three being 2 years old. Weski[15] stated that in 39 people in five generations of one family, 7 men and 9 women had it, while 13 men and 10 women did not. Mirolli[10] reported that in a family of 29, two showed definite indications and 12 showed some symptoms of this deformity.

In almost all of the cases some systemic defect was noted in addition to the hyperplasia. Humphrey[16] cited 10 persons, three of whom were "feeble-minded," two were epileptic, one was deaf, one had "stunted growth" and two had hypertrichosis. Tomes[13] reported that Julia Pastrana, the celebrated "hairy woman," had considerable enlargement of the gums. Waterman[12] mentioned a woman of feeble intellect who was exhibited in Boston under the name of "bear woman," showing huge hypertrophied gums and a remarkable growth of hair over her body. Byars and Sarnat[8] also reported a case with hypertrichosis and "congenital macrogingivae." In such cases an endocrine dysfunction should be considered as a possible etiologic factor.

Histopathologic Findings • Histologic sections of idiopathic fibrous hyperplasia of the gingiva show a moderate hypertrophy of epithelium, with a mild hyperkeratosis, and the production of long rete pegs. The principal cause for the enlargement is in the underlying stroma which shows dense collagen bundles (Fig. 4, left). The fibrous tissue usually is well differentiated, with few young fibroblasts present. Occasional accumulations of chronic inflammatory cells may be present; these are probably caused by local irritation and are incidental findings.

Treatment • Surgical removal of the excess fibrous tissue is the only treatment of value, but the patient should be warned that recurrence of fibrous tissue may follow. Heath[13] reports that Mr. Erickson in 1867 carefully pared off the excess tissue in a child of 2½ years, but

11. Blayney, J. R. Hypertrophic gingivitis. Angle Orthodont. 3:139 (July) 1933.

12. Perkoff, D. Primary generalised hypertrophy of the gums. D Record 49:411 (Aug.) 1929.

13. Heath, C., and Tomes, C. S. Two cases of hypertrophy of the gums and alveoli treated by operation. Brit. J. D. Sci. 22:10, 1879.

14. Murray, J. On three peculiar cases of molluscum fibrosum in children of one family. Medico Chir. Trans. 56:235, 1873.

15. Weski, H. Elephantiasis gingivae hereditarie. Deutsche Monatschr. f. Zahnh. 38:557 (Dec.) 1920.

16. Humphrey, G. M. Unilateral hypertrophy of the gums associated with other abnormalities, chiefly hypertrophical unilateral. Ann. Surg. 3:1, 1886.

the tissue had returned in five years. Ball[9] reported a case in which excess tissue was removed when the child was between the ages of 3 and 5, but the tissue gradually enlarged again.

Newby[17] was forced to extract a tooth for a 19 year old patient with idiopathic hyperplasia of the gingiva, and he noted that the excess gingival tissue disappeared. Consequently, he extracted all of this patient's teeth, and in three months noted an unbelievable reduction in the hyperplastic tissue. He trimmed off the remaining nodules, and constructed complete dentures for the patient. Since the hyperplasia is of the gingival tissue, and since the gingiva disappears when teeth are lost, this observation is not surprising. However, extraction of teeth is not always necessary, since in many instances exposure of the crown of the teeth will allow the underlying teeth to erupt and come into function.

FIBROUS HYPERPLASIA CAUSED BY DIPHENYLHYDANTOIN SODIUM

It is now well known that fibrous hyperplasia of the gingiva often occurs as an undesirable side effect in the use of the anticonvulsant drug, diphenylhydantoin sodium. This drug is more effective in controlling convulsions than the bromides and barbiturates, and does not have the undesirable hypnotic effects, so its use results in an improvement of the general health and mental attitude of the patient. As a result, diphenylhydantoin sodium remains as the drug of choice for use in cases of epilepsy, even though toxic manifestations such as nausea, dizziness, dermatitis, diplopia, or nystagmus may occur.

17. Newby, C. D. A report on a case of hypertrophied gum tissues. J. Canad. D. A. 6:183 (April) 1940.

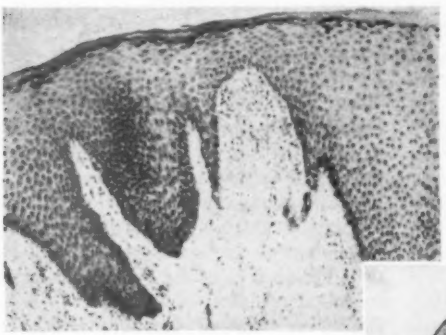


Fig. 5 • Left: Hyperplastic gingival tissue from mouth of 14 year old boy who had been taking diphenylhydantoin sodium for about two years. Note chronic inflammatory reaction around epithelium and the hyperplastic connective tissue

Right: Higher magnification of epithelium shown above. Note well developed hornified layer. (Courtesy of E. D. Coolidge)

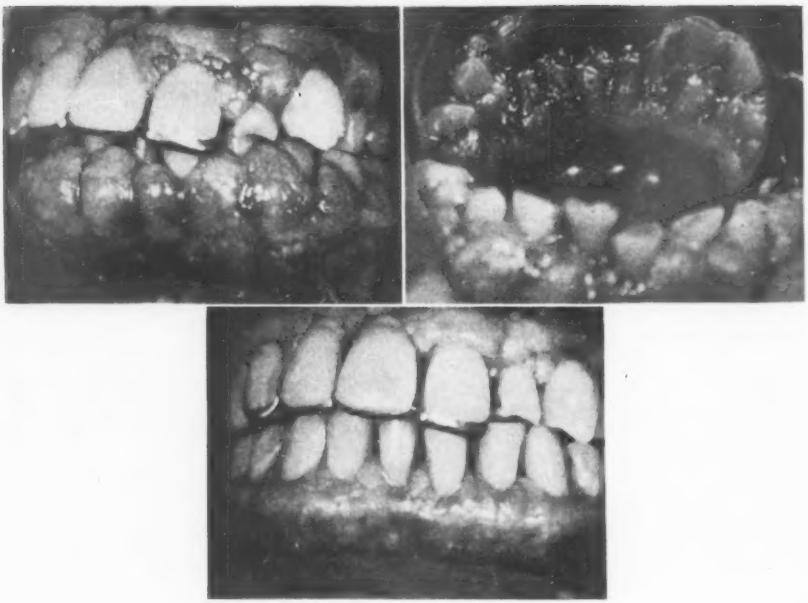


Fig. 6 • Hyperplasia caused by use of diphenylhydantoin sodium in 24 year old patient with epilepsy. Note lobulated gingiva with deep clefts on the labial and lingual aspects. Lower picture was taken a year after gingivectomy which was done for esthetic reasons at patient's request. Note that good home care has prevented much recurrence of the hyperplasia

History • In 1938 Merritt and Putnam[18] reported that diphenylhydantoin sodium was a successful drug for the control of electrically induced convulsions in animals, and soon after this the drug was found to be efficient on persons with epilepsy.[19, 20]

By 1939 reports began appearing that use of this drug caused an overgrowth of fibrous tissue, sometimes to an alarming degree. Apparently only the gingival tissues are involved, since the deformity does not occur in edentulous areas, and nowhere else in the body does the fibrous tissue tend to proliferate. This condition is now well known, since many cases have been reported.

Clinical Description • Gingival changes may begin as early as two weeks after diphenylhydantoin sodium therapy is instituted, but usually no enlargements are noted in the first two or three months. The first change usually is a painless hyperplasia of the gingiva, starting with enlargement of an interdental papilla or two. The surface of the gingiva shows an increased stippling, and finally a "cauliflower" or "warty," granular, or "pebbled" surface (Fig. 4, below). As the enlargement increases, the gingiva becomes lobulated, with clefts remaining

18. Merritt, H. H., and Putnam, T. J. A new series of anticonvulsive drugs tested by experiments on animals. Arch. Neurol. & Psychiat. 39:1003 (May) 1938.

19. Merritt, H. H., and Putnam, T. J. Sodium diphenyl hydantoinate in treatment of convulsive disorders. J.A.M.A. 111:1068 (Sept. 17) 1938.

20. Putnam, T. J. Use of sodium diphenyl hydantoinate as an anticonvulsant. J.A.M.A. 112:2190 (May 27) 1939.

between each enlarged gingiva. Occasionally the enlargement begins as a "marginal festoon" and gradually grows down, covering the crown.

Palpation indicates the tissue is firm, dense, resilient, insensitive, and it shows no tendency to bleed even when severe pressure is used, unless the condition is complicated with a secondary and incidental inflammation caused by local irritation or poor hygiene. Frequently the patient is unaware of the presence of the disease and usually the chief complaint concerns appearance.

Since pronounced hyperplasia will interfere with the maintenance of good oral hygiene, the tissues adjacent to the teeth often will show the usual signs of local irritation—edema, hyperemia or cyanosis, tenderness and tendency to bleed. Removal of the local irritations, when possible, will cause the inflammation to disappear but the fibrous tissue hyperplasia remains.

The location and size of the hyperplasia vary in different mouths, and in different parts of the same mouth. The areas involved most often are the labial surfaces of the upper and lower anterior teeth. Occasionally the growth of tissue will result in movement of the adjacent teeth.[5,21]

Not all patients taking diphenylhydantoin sodium will develop hyperplasia; statistical studies since 1939 report the incidence of hyperplasia variously from 2 to 62.5 per cent.[22]

This wide range can only be explained as representing differences in interpretation by the observers, and differences in the length of time the drug had been administered. Most clinicians now expect hyperplasia to occur in less than half the cases, and in one carefully made study[23] only 10 per cent of 50 cases were severe, and only one case showed an interference with function.

Just why hyperplasia occurs in some but not in all cases when diphenylhydantoin sodium is used is not known. Attempts have been made to correlate the hyperplasia with the size of the daily dosage of the drug, the sex, age, weight or height of the patient, and so forth, but no relation has been established. Apparently the length of time the drug has been taken is of some importance, since the incidence and severity of the hyperplasia is higher in patients who have been under treatment for a long time. Some studies suggested a relationship between vitamin C deficiency, diphenylhydantoin sodium, and the incidence of hyperplasia,[24,25,26] but more recent studies have indicated that vitamin C is not a factor.[27,28] Since none of the findings associated with vitamin C deficiency usually are present (capillary fragility, petechial hemorrhages or gingivitis) it is probable that there is no relation between hyperplasia caused by diphenylhydantoin sodium and avitaminosis C. Another study failed to reveal any dietary deficiency as a basis for the hyperplasia.[28]

Brandon[29] suggested that by-products of diphenylhydantoin sodium are secreted by salivary glands, and cause a proliferation when contacting the gingiva, but offered no evidence to support this thesis. No diminution in the quantity

21. Monier, Leon. Gingivitis hypertrophica. Am. D. Surgeon 47:273 (May) 1927.

22. Reader, Z. A. Gingival hyperplasia resulting from diphenylhydantoin sodium; a review of the literature. J. Oral Surg. 8:25 (Jan.) 1950.

23. Stern, Leo; Eisenbud, Leon, and Klatell, J. Analysis of oral reactions to Dilantin sodium. J. D. Res. 22:157 (April) 1943.

24. Drake, M. E.; Gruber, G. M., and Haury, V. G. The effects of sodium diphenyl hydantoinate on the vitamin C level in tissues and vitamin excretions in rats. J. Pharmacol. & Exper. Therap. 71:268 (March) 1941.

25. Frankel, S. I. Dilantin sodium in the treatment of epilepsy. J.A.M.A. 114:1320 (April 6) 1940.

26. Kimball, O. P. The treatment of epilepsy with sodium diphenyl hydantoinate. J.A.M.A. 112:1244 (April 1) 1939.

27. Burket, L. W. Oral medicine; diagnosis and treatment. Philadelphia, J. B. Lippincott Co., 1948, p. 363-366.

28. Millhon, J. A., and Osterberg, A. E. Relationship between gingival hyperplasia and ascorbic acid in the blood and urine of epileptic patients undergoing treatment with sodium 5-5 diphenyl hydantoinate. J.A.M.A. 29:207 (Feb.) 1942.

29. Brandon, S. A. Treatment of hypertrophy of the gingival tissue caused by Dilantin sodium therapy. J.A.D.A. 37:732 (Dec.) 1948.

of saliva occurs during diphenylhydantoin sodium therapy[23] so the tissues are not dried.

One can conclude that the diphenylhydantoin sodium in some manner predisposes the tissues to gingival hyperplasia, and that local irritations aggravate the condition.

Histopathology • Biopsy studies show nothing dramatic. The stratified squamous epithelium covering the tissue is thick, with a thin keratinized layer. Rete pegs are long and mitotic figures are seldom seen (Fig. 5, left).

In the corium an increase in the size and number of collagen bundles, as well as adult and young fibroblasts are found (Fig. 5, right). Usually areas of chronic inflammation (plasma cells and small round cells) are present, but they are not predominant and probably are a secondary and incidental manifestation. Occasionally areas of bone heteroplasia have been found.[30, 31] No one has reported any evidence that this tissue is malignant.

Treatment • Since the hyperplasia from diphenylhydantoin sodium is painless and in itself harmless to the patient, no treatment is indicated until the deformity is so large that it interferes with mastication, causes a malocclusion or is esthetically objectionable. If the size of the hyperplasia makes treatment desirable, discontinuing the use of the drug will result in a gradual recession of the excess tissue. Most patients, however, prefer to continue the use of the drug, rather than to use a hypnotic. No relation has been shown between the degree of hyperplasia and the amount of diphenylhydantoin sodium taken, but on general principles the dosage of the drug should be as low as is effective.

Mesantoin (methylphenylethyl hydantoin), a new drug sometimes substituted for diphenylhydantoin sodium, apparently will not cause hyperplasia of the gingiva, but this drug reportedly is not as satisfactory an anticonvulsant.[32, 33]

A second method of treatment is to remove the excess tissue surgically. Cautery, whether actual or chemical, is not recommended because of the density of the hyperplastic tissue, but a gingivectomy is easy to perform and the healing is uneventful. The patient should be warned that the deformity may recur (Fig. 6).

If the hyperplasia is not too severe, conservative prophylactic procedures and vigorous home care to maintain good oral hygiene often will produce satisfactory results. In fact, if every patient who is placed on diphenylhydantoin sodium were to follow an effective program consisting of regular dental care and good home care, most cases of diphenylhydantoin sodium hyperplasia could be prevented or kept mild. Stammers and Bromley[34] recommend the use of roentgen ray therapy with a gingivectomy, but this procedure does not seem to be indicated since from the pathologic picture one would expect little benefit.

If all teeth are extracted, the hyperplasia will disappear, but this cannot be recommended unless the teeth are beyond repair.

COMMENT

Just why proliferation of fibrous tissue occurs in one case and not another depends on factors not known. Nor is it known just how the overgrowth of fibrous tissue is formed. Probably there is some proliferation because of irritation to fibroblasts in the area; also often first

30. Thoma, K. H. Oral pathology, ed. 3. St. Louis, C. V. Mosby Co., 1950, p. 574, 1135-1137.

31. Zegarelli, E. V., and Ziskin, D. E. Bone heteroplasia in a case of Dilantin hyperplastic gingivitis. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 29:152 (March) 1943.

32. Aird, R. B. Treatment of epilepsy with methylphenylethyl hydantoinate (Mesantoin). California Med. 68:141 (March) 1948.

33. Malan, G. M., and Harrison, G. G. Mesantoin treatment of epilepsy. South African M. J. 23:516 (June) 1949.

34. Stammers, A. F., and Bromley, J. F. Hypertrophy of the gum associated with epanutin therapy. Brit. D. J. 86:10 (Jan. 7) 1949.

there is an edema present, then a collection of chronic inflammatory cells, including small lymphocytes which can become fibroblasts.[35] The fibroblasts, of course, form the dense bundles of collagen found in fibrous hyperplasia of the gingiva.

Moore[36] states that there is no sharp line of distinction between hyperplasia of connective tissue and neoplasia of fibroblasts, hence there are a number of conditions which resemble fibromas but which should not be so designated. Keloid is one such condition, and fibrous hyperplasia of the gingiva is another. Although these conditions are definitely functionless growths which tend to recur when removed and which resemble fibromas microscopically,[5] they do not show unlimited growth, they may involve one or many gingivae, and their distribution is different from that of a neoplasm. They are not encapsulated and have never been reported to be precancerous.

SUMMARY

1. Fibrous hyperplasia of the gingiva commonly is found in a mild form, and occasionally it can be extensive enough to interfere with eruption and function and hence require surgical removal.

2. The cause of fibrous hyperplasia apparently is local irritation of gingival tissue which has a predisposition to fibrous tissue hyperplasia. Hyperplasia of gingiva will occur in about half of the patients using diphenylhydantoin sodium.

3. Treatment includes surgical removal and the institution of procedures to insure excellent oral hygiene. The enlargements often recur.

4. Fibrous hyperplasia of the gingiva should not be classed as a neoplasm nor be considered precancerous.

35. Maximow, A. A., and Bloom, W. Histology, ed. 4. Philadelphia, W. B. Saunders Co., 1942, p. 113.

36. Moore, R. A. Textbook of pathology. Philadelphia, W. B. Saunders Co., 1944, p. 187.

Hormonal gingivitis

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It has been known for many years that the gingival tissues undergo changes in association with various constitutional states. However, only during the last 15 years or so has more definitive evidence been uncovered to show the relation between endocrine states and gingival disease.

In the study of the gingival histopathology in these conditions the emphasis has been on the epithelial changes. This is readily understood in view of the influence of the work of Papanicolaou[1] and of others who earlier had shown the effects of sex hormones on vaginal epithelium and who had described the variations of the vaginal smear during the sex cycle.

A portion of the author's work cited here has been supported by grants from the Surgeon General's Office, Department of the Army.

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1. Papanicolaou, G. N. Sexual cycle in the human female as revealed by vaginal smears. Am. J. Anat. 52:519 (May) 1933.

CONNECTIVE TISSUE CHANGES CAUSED BY HORMONE ACTION

In this article the influence of some endocrines on connective tissue will be discussed and an attempt will be made to show how alterations in the connective tissue caused by hormone action could explain some clinical disturbances observed in the gingival tissues. This is best introduced by a consideration of some new ideas about connective tissue.

Connective tissue consists of cells, and of extracellular components; namely, fibers, tissue fluid and a ground substance. This ground substance is formed largely of a carbohydrate-protein (glycoprotein) complex which is believed to be organized at the submicroscopic level as a polymer. Gersh and Catchpole[2] have suggested that the degree of polymerization varies depending on the state of the tissue. Thus, loose connective tissue is thought to contain ground substance in a low degree of polymerization, while hard tissues are thought to be characterized by highly polymerized ground substances. Changes in the aggregation of the glycoprotein may contribute to the physical and chemical reactivity of the ground substance.

At the interface between the connective tissue layer and epithelium, in skin, gingiva and in other tissues, the ground substance is altered in a special way. This zone together with the embedded fibers is termed the "basement membrane." The changes which the basement membrane undergoes in physiologic and pathologic states serve as an index of more generalized changes in the adjacent ground substance.

Studies of the ground substance at the microscopic level have received a considerable impetus from (1) the application of freezing-drying fixation and (2) the development of specific histochemical reactions. Through the use of freezing-drying fixation it has been possible to retain constituents which ordinarily might dissolve and diffuse through the tissues. In addition, by this method the denaturation and consequent alteration of tissue proteins is avoided so that further attempts at their characterization in tissue sections can be validly made. The recently developed periodic-acid leucofuchsin reaction[3,4] is highly specific for carbohydrate-containing substances. This reaction, when used following treatment of tissue sections with various buffers, reagents and enzymes, is valuable in the coarse characterization of ground substance.

It can be shown that the major constituents of the glycoprotein ground substance are relatively insoluble in water (unless the tissues are permitted to swell for long periods) under most circumstances. Despite this apparent stability which is characteristic of a highly polymerized structure, the ground substance displays a high degree of lability. In certain states large amounts of water soluble, alcohol insoluble carbohydrate may be extracted from the connective tissue. Thus, with the aid of histochemical technics it is possible to show that in inflammation, in tumors and in connective tissue about growing organs such as the ovary[2] or teeth,[5] the ground substance contains large amounts of these water soluble, alcohol insoluble carbohydrates.

Gersh and Catchpole[2] proposed that these residues arose from enzymatic depolymerization of the highly polymerized glycoproteins of the ground substance, and they postulated that these enzymes were liberated by the cells of the connective tissues. It is possible also that this

2. Gersh, I., and Catchpole, H. R. The organization of ground substance and basement membrane and its significance in tissue injury disease and growth. Am. J. Anat. 85:457 (Nov.) 1949.

3. Hotchkiss, R. Microchemical reaction resulting in the staining of polysaccharide structures in fixed tissue preparations. Arch. Biochem. 16:131 (Jan.) 1948.

4. McManus, J. F. A. Histological demonstration of mucin after periodic acid. Nature 158:202, 1946.

5. Engel, M. B. Some changes in the connective tissue ground substance associated with the eruption of the teeth. J. D. Res. 30:322 (June) 1951.

water soluble material represents an altered secretory activity of the connective tissue cells in some states.

It has become increasingly clear that these changes in connective tissue can occur in response to hormone influence. Two striking examples which may be cited occur in the cases of the guinea pig pubic symphysis[6] and in the sex skin of the monkey.[7] In these instances it has been possible to define the nature of the connective tissue changes in somewhat different terms. The implications of this work may be extended to connective tissue generally and to the gingiva specifically.

Physicochemical methods and theory have been employed in studying the changes in the ground substance of the pubic symphysis of the guinea pig as the symphysis relaxes in pregnancy or under the influence of the hormone-like substance, relaxin.[8] It was concluded that the connective tissue behaves in such a manner as to indicate the presence of negatively charged immobile macromolecules. As the symphysis relaxes preceding parturition it appears that the density of these negatively charged aggregates is reduced. Because of the presence of acidic groups in such compounds as hyaluronic acid and chondroitin sulfuric acid in the ground substance glycoprotein, one might expect these large molecules to display negative charges at physiologic pH.

Similar changes can be induced in sex skin with some hormones. Under the influence of gonadotrophic hormones, acting through the ovary, or of estrogen administered directly, the sex skin in the monkey shows a clear response. The skin swells in the course of a few days. Electrochemical studies of this tissue have been made which show that with the swelling the density of negatively charged colloid is reduced. Preliminary studies on the gingiva of these animals show that they follow a similar course. This interpretation is supported by histochemical study of the sex skin. Administration of the sex hormones (estradiol and gonadotrophin) leads to an increase in the amount of alcohol insoluble, water soluble carbohydrate. The basement membrane is attenuated and the demonstrable ground substance is reduced.

The electrochemical studies reveal a difference in the interaction of ground substance colloid with potassium and sodium ions. While potassium mobility is greater than sodium in water, this situation tends to become reversed in highly polymerized ground substance, and potassium ion relative mobility then bears an inverse relation to the degree of aggregation of the ground substance. The selective reaction with cations implicates the connective tissue in a fundamental role in electrolyte and water metabolism.

As a result of these studies of endocrine influences the presence of a heterogeneous system in the ground substance has been postulated. That is, the coexistence of at least two phases which are in equilibrium is presumed. One of these is a colloid-rich, water-poor phase, the other is water-rich, colloid poor (the glycoprotein of ground substance is here designated "colloid"). In solid fibrosed states the colloid-rich phase is thought to predominate while in edema or in boggy tissues the water-rich phase is believed to form the larger component. The uptake or loss of water and salts by tissues could be regulated by an exchange between the two phases without affecting the cells or the blood. This would lead to changes in the relative quantities of the phases but the composition of each of the phases would remain constant. Through this arrangement the connective tissue of the

6. Perl, E., and Catchpole, H. R. Changes induced in the connective tissue of the public symphysis of the guinea pig with estrogen and relaxin. Arch. Path. 50:233 (Aug.) 1950.

7. Duran-Reynals, F.; Bunting, H., and von Wagenen, G. Studies of the sex skin of macaca mulatta. Ann. New York Acad. Sci. 52:1006 (May) 1950.

8. Joseph, N. R.; Engel, M. B., and Catchpole, H. R. Interaction of ions and connective tissue. Biochimica et Biophysica Acta. In press.

skin and of the gingiva are thought to contribute to the maintenance of homeostasis.

Through histochemical and analytical studies it has become clear that in many "hyperplastic" or inflammatory states, and in some degenerative conditions, the gingiva contains large amounts of water soluble mucoprotein carbohydrate which is conceived to represent excessive amounts of water-rich colloid-poor phase. Thus in normal gingiva, when the mucoprotein is extracted and estimated, one finds an average of 250 mg. of carbohydrate per 100 Gm. of dry tissue. In inflamed gingiva the average value is around 400 mg. per 100 Gm.

These preliminary considerations lead to a somewhat more rational understanding of the clinical entities which will be discussed.

PREGNANCY GINGIVITIS

During pregnancy the entire endocrine system is involved. There is evidence of heightened secretion of steroid hormones by the ovaries, adrenal glands and placenta, and of increased production of anterior pituitary-like hormones. In later stages, the relaxation hormone also may be present.

In view of the effects of hormones on connective tissue it is not surprising that in some women there are gingival disturbances during pregnancy. There are a number of comprehensive reviews of this problem together with original material.[9-13] The clinical condition may vary from a localized inflammation of the gingival margin, particularly about the anterior teeth, to generalized involvement of the interdental papillae and of the free gingival margin. Occasionally the swelling of individual papillae may be so extensive as to give rise to the so-called "pregnancy tumor." Many inflammatory swellings which may have been present before pregnancy have a tendency to develop further during this period.

The characteristic findings reported after histopathologic examination are hyperplasia of the epithelium in the stratum germinativum and degenerative changes in the stratum spinosum with pearl formation. The connective tissue changes described are similar to those seen in inflammation. In "pregnancy tumors" the characteristic connective tissue change centers about increased capillary proliferation.

Some clinicians recommend surgical treatment of the larger masses such as the so-called "pregnancy tumors." Others point out that these may regress and disappear without such drastic treatment. While pregnancy does lead to definite changes, there is a considerable body of opinion that favors the view that in a normal mouth where hygiene is good this condition will not develop. The logical development of such a view is that local treatment can restore a normal tissue state. However, there is considerable evidence that pregnancy can induce primary changes in tissues, including gingival tissue, independent of initial lowering of tissue resistance. Local treatment or maintenance of conscientious home care are beneficial. Following parturition and the resumption of normal hormonal levels, there usually is a regression of the gingival changes.

CYCLICAL CHANGES IN THE GINGIVA

Similar effects to those observed in pregnancy are observed during the female sex cycle. Thus swelling, edema and reddening of the gingival margin occur

9. Blum, T. Pregnancy tumors. J.A.D.A. 18:393 (March) 1931.

10. Maier, A. W., and Orban, B. Gingivitis in pregnancy. Oral Surg., Oral Med. & Oral Path. 2:334 (March) 1949.

11. Monash, S. Proliferative gingivitis of pregnancy. D. Items Interest 48:500 (July) 1926.

12. Ziskin, D. E.; Blackberg, S. N., and Stout, A. P. The gingivae during pregnancy. Surg. Gynec. & Obst. 57:719 (Dec.) 1933.

13. Ziskin, D. E., and Nesse, G. J. Pregnancy gingivitis; history, classification, etiology. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 32:390 (June) 1946.

in the period just preceding menstruation. Gingival bleeding is elicited easily or even may occur spontaneously. Following menstruation there is a regression of these changes. Apthous lesions in the oral cavity may occur concurrently. Reports have appeared describing the coexistence of similar lesions in the oral cavity and in the vagina.[14,15,16] Recent attempts to establish a cyclical variation in epithelial smears from the oral cavity have not been successful.[17,18]

While the hormonal background of these disturbances is assured, it is not at all clear which hormones are involved. If these lesions are considered homologous to the reactions of the endometrium they may represent a heightened reactivity of the connective tissue to estrogen withdrawal or to a change in the estradiol-progesterone ratio. The precise etiology of these cyclical changes can become known only as the physiology of menstruation becomes better understood.

Gynecologists have treated patients with apthous lesions by using anterior pituitary extracts which contain gonadotrophins. Since the exact hormonal mechanisms remain obscure in these cyclical gingival changes it would appear that they are best treated by local supportive measures.

PUBERTAL GINGIVITIS

During the pubertal period it is not uncommon to observe gingival changes in both boys and girls. A generalized gingival hypertrophy may result. The interdental papillae are swollen and boggy. A number of such cases have been reported in detail by Coolidge[19] in his study of hypertrophic gingivitis. While this condition may be excited by the poor oral hygiene of the hasty adolescent the predisposing factor undoubtedly is the hormonal state. In patients of this group orthodontic treatment frequently excites a hypertrophic response which previously was mild or even absent. The histopathologic changes which have been reported are essentially those characteristic of chronic inflammation and they are similar to those noted in pregnancy or menstruation. Treatment in these cases depends on local measures such as removal of local irritations from calculus or fillings and the restoration of oral hygiene. Mild astringents may be of value. The condition is transient and disappears following the obscure endocrine adjustment which occurs in adolescence.

CHRONIC ATROPHIC SENILE GINGIVITIS

Atrophic changes may develop in the oral mucosa and particularly in the gingiva following the menopause. The tissues become pale and anemic and grayish white patches may appear. The patients complain of dryness and a burning sensation. There may be gingival bleeding. Similar changes sometimes occur in the genital tract.

Histopathologic examinations show beginning atrophy of the prickle cell layer with occasional early keratinization of the corneum and there may be varying stages of leukoplakia.[20] Connective tissue changes have not been described.

Since the climacteric is associated with diminished production of estrogen it has been assumed that absence of these hormones is responsible for the gingival dis-

14. Jones, O. V. Cyclical ulcerative vulvitis and stomatitis. J. Obs. and Gyn. of the British Empire 47:557 (Oct.) 1940.

15. Nathanson, I. T., and Weisberger, D. B. Treatment of leucoplakia buccalis and related lesions with estrogenic hormone. New England J. Med. 221:556 (Oct.) 1939.

16. Ziserman, A. J. Ulcerative vulvitis and stomatitis of endocrine origin. J.A.M.A. 104:826 (March 9) 1935.

17. Montgomery, P. W. A study of exfoliative cytology of normal human oral mucosa. J. D. Res. 30:12 (Feb.) 1951.

18. Papic, M., and Glickman, I. Keratinization of human gingiva in menstrual cycle and menopause. Oral Surg., Oral Med. & Oral Path. 3:504 (April) 1950.

19. Coolidge, E. D. Hypertrophic gingivitis. J.A.D.A. 28:1381 (Sept.) 1941.

20. Richman, M. J., and Abarbanel, A. R. Effects of estradiol, testosterone, diethylstilbestrol and several of their derivatives upon the human oral mucous membrane. J.A.D.A. 30:913 (June) 1943.

turbances. Accordingly, the parenteral administration of estradiol and diethyl stilbestrol and the use of these substances in ointments have been recommended.[15,20] Reversal of atrophic changes, restoration of the gingiva to their normal pink color, and disappearance of subjective symptoms have been reported.

CHRONIC DESQUAMATIVE GINGIVITIS

A desquamative type of gingivitis is encountered occasionally.[21,22,23] The gingiva is covered by multiple raw, bleeding areas. The epithelium is lifted easily from the underlying connective tissue; it may peel spontaneously. The lesions may begin as vesicular eruptions which break, leaving raw connective tissue surfaces exposed. While some writers claim that the lesions do not occur in edentulous areas this is not a constant observation. During acute phases the cervical lymph nodes may be enlarged.

The lesions cause a good deal of discomfort and soreness. While there are periods of remission, these are short and incomplete and the disturbance may persist for many years. This condition occurs predominantly in women, although men are sometimes affected. In most instances the onset is around the menopause, and other pathologic conditions, such as lichen planus and breast tumors, have been found in association with the gingival disturbance. Ziskin and Zegarelli[23] were among the first to suggest that some dysfunction involving the sex hormones was responsible.

As a result of studies conducted in the laboratory of the University of Illinois College of Dentistry[21] it has been shown that profound changes occur in the connective tissue ground substance in these cases. The basement membrane either is absent or attenuated and the ground substance is believed to be depolymerized as suggested by its changed reactivity with the periodic-acid leucofuchsin stain. Abnormally large amounts of alcohol insoluble, water soluble carbohydrate may be extracted. The glycoprotein cementing substance between the epithelial cells also is affected. This, together with the basement membrane alteration, probably contributes to the ready loss of the epithelial layer. It has been suggested that these changes may be caused by the production of increased amounts of depolymerizing enzymes in the connective tissue. It is also possible that there is a failure in the synthesis of ground substance, such that the water-rich, colloid-poor phase predominates.

A treatment, either by local application or by injection of estrogenic hormones, has been suggested.[23] However, these measures are not always of value. Surgical removal of the tissue results in a temporary improvement at the site of excision. Subsequently the lesions may recur. Treatment with antibiotics has been unsuccessful although such measures may reduce the secondary infection. On the whole this condition does not respond well to any known measures.

SUMMARY

In many of the conditions mentioned, such as pregnancy and puberty, the endocrine activity represents a physiologic alteration. The organism is making a transition and during the period of adjustment there are transient symptoms in target tissues. While the connective tissue changes in all of these conditions have not been completely investigated, it appears that they are closely related (with the exception of atrophic gingivitis). This is not to say that the same hormone or hormones are involved in each instance,

21. Engel, M. B.; Ray, H. G., and Orban, B. The pathogenesis of desquamative gingivitis. J. D. Res. 29:410 (Aug.) 1950.

22. Sorrin, S. Chronic desquamative gingivitis. J.A.D.A. 27:250 (Feb.) 1940.

23. Ziskin, D. E., and Zegarelli, E. Clinical and histopathologic study of chronic desquamative gingivitis and its treatment with the sex hormones. (Abst.) J. D. Res. 21:341 (June) 1942.

but rather that the range of response in connective tissue to any stimulus is necessarily circumscribed. However, certain inherent qualities of the tissues at different periods may affect the manner in which they respond to hormones.

In pregnancy gingivitis, in pubertal gingivitis, in gingivitis associated with menstruation, as well as in the extreme case of desquamative gingivitis, there are abnormal amounts of water soluble, alcohol insoluble glycoprotein residues in the tissues. This may reflect either disturbances in the synthesis of ground substance or increased activity of depolymerizing enzymes, or both. Shifts in the water and electrolyte metabolism are implicit in these changes of ground substance.

CLINICAL USE OF HORMONES IN TREATMENT OF GINGIVAL DISEASE

From both animal experiments and clinical trial in human patients it has been shown that hormones exert an influence on the state of the gingival tissues. The work of Ziskin and his collaborators with estrogens and with some gonadotrophins has shown the possibility, at least for clinical improvement, with the former, particularly in desquamative gingivitis. The use of estrogens in atrophic gingivitis also appears promising. However, the finding of Ziskin and co-workers[24,25] that some gonadotrophins apparently cause undesirable gingival changes should not be overlooked.

While there is considerable literature dealing with the gingival symptoms caused by endocrine disturbance, there are relatively few reliable reports on the matter of endocrine therapy. It is to be expected that with the tremendous impetus given to endocrinology by the investigations on the steroid hormones, the effect will radiate to oral medicine. A thoughtful person is forced to conclude that endocrine therapy, even in the practice of medicine, despite some notable exceptions, remains in many respects almost empirical, if not dangerous. The secondary effects of a hormone may be more undesirable than the apparent ameliorative action. This has been shown many times in the case of cortisone. With the sex steroids the possible carcinogenic effects cannot be ignored.

In many instances the question is not simply one of hormone insufficiency or excess. The interrelations between the endocrine glands is complex and incompletely understood. Many of the effects on the target tissues remain unknown. It would seem prudent, therefore, to recommend consultation with an endocrinologist in those cases of persistent hormonal gingivitis where hormone treatment appears to be the only solution.

24. Ziskin, D. E.; Blackberg, S. N., and Slanetz, C. A. Effects of subcutaneous injections of estrogenic and gonadotrophic hormones on gums and oral mucous membranes of normal and castrated rhesus monkeys. J. D. Res. 15:407 (Dec.) 1936.

25. Ziskin, D. E. Effects of certain hormones on gingival and oral mucous membranes. J.A.D.A. 25:422 (March) 1938.

Knowledge • If you can measure that of which you speak, and can express it by a number, you know something of your subject: but if you cannot measure it, your knowledge is meager and unsatisfactory. *Lord Kelvin.*

Oral manifestations of the blood diseases

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Some blood diseases have obvious effects on gingival and periodontal tissues.

Although each of the blood dyscrasias is an entity, certain basic changes in the blood recur frequently in various conditions. Anemia, leukopenia and thrombocytopenia are observed in many disorders of the hemopoietic tissues and, whenever present, are likely to produce the same signs and symptoms.[1] Symptoms in the mouth often are the earliest signs of the disease and the patient will seek aid of the dentist first. Pallor and decreased reparative powers are attributable to the anemia; ulceration of mucous membranes and apical abscess to the leukopenia; petechiae and bleeding from the mucous membranes to the thrombocytopenia.[2]

The blood dyscrasias generally are considered in connection with diseases of the white blood cells, diseases of the red blood cells, and hemorrhagic diseases.

DISEASES OF THE WHITE BLOOD CELLS

Leukemia • Leukemia is a fatal disease in which there is considerable increase in the number of white blood cells as a result of a "neoplastic proliferation" of the leukoblastic tissues.[3]

The disease is widely distributed; no race or groups of people are immune. The disease also has been described in many species of animals. Weinmann[4] has shown a leukemic infiltration of the periodontal tissues of rat molars.

The disease may occur at any age and in either sex. However, the various types appear at different ages. An acute leukemia is most prominent in the first five years. Chronic myelogenous leukemia is most common in the decade between 25 to 35 years of age; chronic lymphatic leukemia between the ages of 40 and 60.

Changes of the oral and gingival tissues in these diseases have been noted and clinical variations have been described according to the type of leukemia.

Clinical Studies • In acute leukemia the first manifestations are in the head and neck. These manifestations are aching of teeth without caries; enlargement of submaxillary, submental and cervical nodes occurring especially in monocytic and lymphatic nodes, and swelling of lips and face.

The gingival picture is one of necrosis, ulceration, spontaneous hemorrhage and hyperplasia. Gingivitis and stomatitis are among the earliest symptoms of acute leukemia.[5]

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2. Moore, C. V. Hematologic aspects. In University of Illinois manual of telephone extension course, 1950-1951. Current advances in dentistry. Chicago, University of Illinois College of Dentistry, 1951.

3. Thoma, K. H. Oral pathology, ed. 2. St. Louis, C. V. Mosby Co., 1944.

4. Weinmann, J. P. Personal communication.

5. Moloney, W. C. Clinical significance of oral lesions in acute leukemia. New England J. Med. 222:577 (April) 1940.

Forkner,[6] Burket,[7] Love,[8] Cook,[9] Mallett and Guralnick,[10] Armbrecht and Apple,[11] and Saghirian and Jones[12] have compiled data on the oral symptomatology.

Histologic Studies • The altered blood formation is reflected in the gingiva and periodontal tissues. In acute myeloid leukemia the normal mature leukocytes in a simple gingivitis are replaced by immature leukocytes. These cells arise in the bone marrow where altered hemopoiesis is occurring, and after circulation in the blood, migrate to areas of inflammation, in this instance the gingiva. Weinmann[4] suggests this should be termed a leukemic gingivitis.

Burket[13] has endeavored to correlate the histopathologic changes and clinical symptoms of acute leukemia. He related the necrosis to the thrombosis of blood vessels, and the tissue enlargement to extravascular accumulation of abnormal cells. Karcher and McCarthy[14] also describe a heavy infiltration of the lamina propria with monocytic cells in a case of acute monocytic leukemia.

De Aprile[15] referred to the histologic picture in the gingiva as a granuloma, and observed that, similar to scurvy, there was no tendency for organization of the fibrous connective tissue. Berberich[16] concurred with de Aprile and refers to a scurvy-like character of the lesions and to the infiltrations which grossly give the tissue the aspect of a tumor.

Mason[17] and Thoma[3] describe an infiltration of cells in the gingiva with immature cells characteristic for the type of disease predominating.

Wentz[18] studied the case histories of 149 patients in relation to the oral findings. Pallor, hyperplasia of the gingiva, spontaneous hemorrhage, petechiae and ulceration showed a higher incidence in the acute than in the chronic forms. Biopsy specimens of 13 patients were studied. A diagnosis could be made in those patients with acute myelogenous leukemia. An infiltration of immature myeloid elements was noted. Some of the immature cells exhibited mitotic activity. A pocket smear showed similar immature cells. No characteristic gingival changes were noted in patients with chronic myelogenous and lymphatic leukemia.

Agranulocytosis • The disease is characterized by reduction in the number of granulocytes in the peripheral blood. The other blood elements are not affected.

Gingivitis and stomatitis are both frequent and early symptoms of acute agranulocytosis. Histologic sections of the gingival tissue in this disease have been studied in only a few cases. In a case of agranulocytosis[19] with aplasia of the granulocytic elements in the bone marrow, histologic examination of a gingival papilla showed a lack of keratinization of the epithelium and edema of the deeper epithelial layers, and edema and hyperemia of the subepithelial con-

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9. Cook, T. J. Blood dyscrasias as related to periodontal disease with special reference to leukemia. J. Periodont. 18:159 (Oct.) 1947.

10. Mallett, S. D., and Guralnick, W. C. A case of acute monocytic leukemia. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 28:95 (Feb.) 1942.

11. Armbrecht, E. C., and Apple, C. W. Lymphatic leukemia. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 28:607 (Oct.) 1942.

12. Saghirian, L. M., and Jones, C. A. Acute monocytic leukemia. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 28:561 (Oct.) 1942.

13. Burket, L. W. Histopathologic explanation for the oral lesions in the acute leukemias. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 30:516 (Sept.) 1944.

14. McCarthy, F. P., and Karcher, P. H. The oral lesions of monocytic leukemia. New England J. Med. 234:787 (June 13) 1946.

15. De Aprile, E. Carames. Sobre histopatologia de la gingivitis. Buenos Aires, Argentine, Amorrortu, 1945.

16. Berberich, J. Lehrbuch der Mund und rachen Krankheiten. Leipzig, Germany, G. Thieme, 1932.

17. Mason, H. Gingival hypertrophy due to myelogenous leukemia. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 28:738 (Dec.) 1942.

18. Wentz, F. M.; Anday, D. J., and Orban, B. Histopathologic changes in the gingiva in leukemia. J. Periodont. 20:119 (Oct.) 1949.

19. Anday, D. J., and Orban, B. Acute agranulocytosis. Arch. Path. 39:369 (June) 1945.

nective tissue. The narrow zone surrounding the necrotic area was infiltrated by plasma cells, macrophages, and lymphocytes. Neutrophilic leukocytes were absent. Many of the plasma cells were binucleated and trinucleated. The capillary endothelial cells were swollen and migration from the capillaries was reduced to a few lymphocytes and macrocytes.

The lesions in the oral cavity usually begin with a hemorrhagic cyanotic area which becomes sharply demarcated, necrotic, and soon ulcerates. The ulcer is covered by a dirty yellow gray or greenish black membrane. This appearance suggests there is a breaking down of the tissue because of the absence of neutrophils.[1]

DISEASES OF THE RED BLOOD CELLS

Anemia • Pallor is the most striking symptom of anemia. A patient with normal coloration in the buccal and pharyngeal mucous membranes and conjunctivae rarely will be found to be anemic.

Pernicious Anemia • Pernicious anemia is a deficiency disease caused by a lack of specific substance necessary for growth and development of the erythrocyte. The characteristic sore tongue or glossitis may be its earliest symptom and may appear when there is little or no anemia.[20] After several attacks of glossitis the epithelium of the tongue atrophies and the papilla disappear and the smooth glazed tongue characteristic of pernicious anemia remains.[1]

Polycythemia • In direct comparison to the anemias, this is a disease characterized not by too few but by too many red cells which may number 8,000,000 per cu.mm. or more. The patient shows a dusky deep red color of the mucous membrane together with a hemorrhagic tendency, the cause of which is often unknown.[2]

HEMORRHAGIC DISEASES

Hemorrhage into or from the mucous membranes or gingiva may be the first sign of disorder of the hemopoietic organs. In general the mechanism of bleeding results from one or more of the following: (1) Quantitative or qualitative changes in the platelets; (2) disturbances in clotting mechanism, and (3) increased permeability of capillary walls.[2]

Thrombocytopenic Purpura • This is a symptom complex manifested by hemorrhages into the skin and from mucous membranes and gingiva. Idiopathic thrombocytopenic purpura occurs most frequently in children, and the female sex.

The hemorrhages are associated with a reduced platelet count, prolonged bleeding time and a normal coagulation time. The clot is nonretractile.

Usually the bone marrow shows adequate megakaryocytes, a finding which in most instances assumes a satisfactory platelet response following splenectomy.

Hemophilia • In hemophilia the symptoms depend on a defect in the coagulation mechanism. It is a hereditary disease characterized by the transmission of a sex-linked recessive Mendelian defect characterized by recurring hemorrhages throughout life. The disease almost always occurs in the male, and is transmitted by females. The most frequent sites of hemorrhage are the mouth, lips, gingiva, and tongue. Except for prolonged coagulation time, all blood studies are normal.

Bleeding resulting from increased capillary permeability in which the platelet count is normal is observed in such chronic diseases as nephritis, intoxications and scurvy.

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CONCLUSIONS

Diseases of the hemopoietic organs often are manifested by local signs and symptoms in the gingiva, periodontal tissues and oral cavity. In all these diseases characteristic pathologic changes will occur not only in the periodontal tissues but also in other organs, and since this is true, it is not possible to compare these periodontal symptoms of a systemic disease with other periodontal diseases.[21]

In acute leukemia, a myeloid or monocytoid infiltration is a gingival manifestation of systemic disease and is specific in that a diagnosis could be made from the biopsy alone on the recognition of the characteristic immature leukocytes.[18]

21. Orban, B. Symposium on systemic aspects of periodontal diseases. J. Periodont. 19:123 (Oct.) 1948.

Periodontitis: etiology, pathology, symptomatology

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The term "periodontitis" designates the pathologic changes of the periodontal tissues, inflammatory in character. These changes involve the gingiva, the cementum, the periodontal membrane, the alveolar bone and the alveolar process. Periodontitis can be classified from a topographical point of view according to the initial attack as (1) apical periodontitis and (2) marginal periodontitis. Marginal periodontitis is termed by many "periodontitis simplex," to differentiate it from "periodontitis complex." The latter is a secondary inflammation following the degenerative changes of periodontosis. Periodontitis simplex, or marginal periodontitis, on the other hand, develops as a deeper penetration of a gingivitis. Marginal periodontitis should not be confused with the clinical and pathologic picture of horizontal atrophy that is of a noninflammatory, regressive character. The present discussion will be limited to marginal periodontitis.

With this definition in mind it is possible to scrutinize and evaluate the opinions of different authors on this subject, although these may be hidden under such ill-defined terms as "periodontoclasia," "pericementitis," or "alveolar pyorrhea."

ETIOLOGY

Marginal periodontitis, by definition, is the inflammatory reaction of the periodontal tissues to any kind of irritation.

Although a hematogenous propagation of microorganisms into the periodontal tissue—for example, of tubercle bacilli—has been observed, the blood-borne infection of the periodontal tissues is so rare that hematogenous periodontitis can be omitted from this discussion. The overwhelming majority of cases of periodontitis are initiated by an irritation of the gingival surface resulting first in a gingivitis. Therefore the etiologic factors for gingivitis and marginal periodontitis

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are the same. The inflammation in the periodontal tissue can be caused by physical and chemical irritants or by biotic irritants.

The severity and the degree of progress of the inflammation from the gingiva into the supporting tissue apparently depend not only on the intensity of the irritation but also on the resistance of the periodontal tissues. The normal resistance of the periodontal tissues may be diminished by systemic factors—for example, by nutritional deficiencies or hormonal disturbances; by local factors such as occlusal trauma, or by degenerative diseases of the periodontal tissues such as periodontosis.

Physical Irritants • It is generally accepted that impaction of food, caused through neglect of oral hygiene or through loss of contact between two teeth, crowding of teeth, proximal carious lesions or overhanging margins of fillings and crowns, will lead to gingivitis and marginal periodontitis.

Deposition of calcium salts in materia alba which leads to formation of calculus deposits is one of the most frequent causes of inflammatory periodontal diseases.

Biotic Irritants • Barrett,[1,2] in 1914, found *Endamoeba gingivalis* in periodontal pockets of 46 patients but was unable to find them in seven individuals with normal periodontal tissues. His findings were confirmed by Bass and Johns[3] and by Kofoid.[4] However, Dobell[5] has stated that there was not sufficient evidence that the *Endamoeba gingivalis* caused marginal periodontitis.

Miller[6] isolated 22 different bacterial species from 27 cases of marginal periodontitis, identifying only *Staphylococcus albus* and *Staph. aureus*. Kirkpatrick[7] found pneumococci in lateral abscesses. Goadby[8] and many others frequently cultivated the following organisms: staphylococci, streptococci of many types, pneumococci, members of the genus *Neisseria, Gaffkya tetragena, Bacillus cloacae,* coliform bacilli, diphtheroids, lactobacilli, leptothrices and yeasts.

A number of investigators found spirochetes, with or without fusiform bacilli, in periodontal lesions. Present-day knowledge of the bacteriology of periodontal diseases is summarized by Appleton,[9] who reviews the literature exhaustively. He states that the results of bacteriologic studies "indicate that no particular species can be incriminated as the specific etiologic factor and that in most, if not all, cases we are dealing with a mixed infection."

Further investigations are needed to decide whether microorganisms enter and can survive in living tissues. Most investigators are of the opinion that the development of marginal periodontitis is caused by the toxins produced by the organisms thriving on the surface of the gingiva and in the gingival sulcus and pocket and not by an invasion of bacteria into the tissues. Fish[10] believes that organisms may enter the periodontal tissues through an ulcer at the bottom of a pocket. Such bacteria either are destroyed by the defense cells of the tissues or they survive and cause an acute lateral abscess.

Systemic Factors • Clinical experience shows that severe local irritation of the

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2. Barrett, M. T. Clinical report upon amoebic pyorrhea. D. Cosmos 56:1345 (Dec.) 1914.
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5. Dobell, Clifford. The amoebae living in man. New York, William Wood & Co., 1920.
6. Miller, W. D. Die Mikroorganismen der Mundhoehle, ed. 2. Leipzig, G. Thieme, 1892.
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9. Appleton, J. L. T. Bacterial infection, ed. 4. Philadelphia, Lea & Febiger, 1950, p. 509.
10. Fish, E. W. Surgical pathology of the mouth. Philadelphia, J. B. Lippincott Co., 1951.

gingival tissues can persist over a long period of time without the inflammation penetrating into the depths and producing the symptoms of marginal periodontitis. Such observations led to the postulate of additional factors in the pathogenesis of marginal periodontitis, such as systemic and hereditary factors. Dietary and hormonal disbalances have been considered most frequently in this connection. However, a critical evaluation of the available reports shows that the significance of specific systemic factors as etiologic components in the pathogenesis of marginal periodontitis has not been established.

Local Factors • No experimental evidence is available for the claim that traumatic occlusion by itself leads to the development of marginal periodontitis. It is, however, certain that occlusal imbalance plays a contributing and aggravating role in the progress of an initial inflammatory reaction. Occlusal trauma leads to severe injury or even to a necrosis of parts of the periodontal membrane and the damaged tissue is, of course, less resistant to local irritant factors.

Degenerative Diseases of the Periodontal Tissues • Clinical experience and histologic investigations[11, 12] demonstrate that periodontosis, after the first mainly degenerative stages, is soon complicated by periodontitis. The formation of deep pockets, a consequence of the degeneration of the connective tissue and the proliferation of the epithelium, leads to accumulation of irritant debris and bacteria and consequently to inflammatory reaction.

PATHOLOGY

The differential diagnosis of gingivitis or marginal periodontitis is made chiefly on clinical grounds and, therefore, is often made arbitrarily. Histologic studies reveal that the deeper supporting structures show reactive changes even in slight cases of gingivitis. On the other hand it is obvious that all cases of marginal periodontitis show inflammatory reaction of the gingiva. The pathology of gingivitis has been treated in another report.[13] The present discussion deals with the progress of gingival inflammation into the supporting structures of the teeth[12] and the changes in the alveolar bone, the periodontal membrane and the cementum.

One group of investigators[14-18] claims that the inflammation of the gingiva proceeds directly into the periodontal membrane. Another group[19, 20, 21] endorses the same concept as the first with the reservation that, occasionally, the inflammation extends to the external surface of the alveolar process or into the bone marrow spaces. Only a few authors[22, 23, 24] assert that the inflammation passes from the gingiva into the alveolar bone, involving the bone marrow and later the periodontal membrane.

In order to decide the crucial question of the propagation of gingival inflam-

11. Orban, B. Zahnfleischtasche und Epithelansatz. Ztschr. f. Stomatol. 29:859 (July); 29:1005 (Aug.); 29: 1359 (Dec.) 1931.

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22. Thoma, K. H., and Goldman, H. M. Classification and histopathology of parodontal disease. J.A.D.A. 24:1915 (Dec.) 1937.

23. Goldman, H. M. Periodontia, ed. 2. St. Louis, C. V. Mosby Co., 1949.

24. James, W. W., and Counsell, A. Histological investigation into so-called pyorrhea alveolaris. Brit. D. J. (Oct. 15) 1927.

mation, 15 human jaws from persons ranging in age from 11 to 74 years were studied histologically.[12] The investigation showed that the invasion of the periodontal membrane is a late and secondary phenomenon. As a rule the inflammation progresses either to one of the periosteal surfaces of the alveolar bone or into the marrow spaces of the interdental septum. Only after the destruction of the bone separating the inflammatory focus from the periodontal membrane proper, is the latter involved. At the same time, the periodontal membrane ceases to exist in the strict sense of the word.

The explanation for this behavior of the inflammatory process is simple. An almost invariable rule for the propagation of inflammations is that they follow the path of least resistance. The ligamentous character of the normal periodontal membrane is an efficient block to the progress of any gingivitis. In the case of marginal gingivitis the progress is often into the attached gingiva; that is, to the vestibular or oral periosteal surface of the alveolar process. The loose connective tissue between the fiber bundles of the attached gingiva offers much less resistance than does the much denser periodontal membrane.

Papillary gingivitis shows an even more interesting mode of penetration into the deeper tissues. The terminal branches of the interdental artery, vein, lymph vessels and nerves enter the interdental papillae. These structures are designed principally for blood and nerve supply, and for blood and lymph drainage of the periodontal membrane of two adjacent teeth. This bundle of structures is surrounded by loose connective tissue and is contained in the interdental canals.[25, 26] The papillary gingivitis spreads into these canals, involving secondarily adjacent marrow spaces in the interdental septum. Only after destruction of the alveolar bone proper from the rear, as it were, can the periodontal membrane be invaded and destroyed.

A primary involvement of the periodontal tissue, or better, a direct invasion of the periodontal space, can occur if the periodontal membrane is damaged and consequently has lost its ligamentous character. This may happen either in periodontosis, where the degenerative process leads to a replacement of the alveolodental ligament by loose connective tissue, or in occlusal trauma where hemorrhages in, or necrosis of, the marginal parts of the periodontal membrane occur. Finally, in rare instances, the inflammation, after invading the marrow spaces, spreads along one of the horizontal branches of the interdental vessels and nerves into the periodontal membrane.

Though evidently there is no sharp demarcation between gingivitis and marginal periodontitis, it is clear that the entire depth of the gingiva has to be involved before a marginal periodontitis can develop. During this process the gingival and the transseptal fibers that are anchored in the cementum are detached from the tooth, disappear and are replaced by inflamed granulation tissue. At the same time the epithelial attachment proliferates along the root surface, while in the more coronal parts it is separated from the tooth; thus the pocket deepens and its depth is a clinical measure of the progress of the destruction.

The mechanism of pocket formation, especially that of epithelial downgrowth, is still controversial. Some authors believe that the process of detachment and destruction of the gingival fibers is primary, the downgrowth of the epithelium, secondary. Others maintain that the proliferation of the epithelial attachment is primary and comparable to

25. Zuckerkandl, E. Handbuch der Zahnheilkunde, I. Vienna, Hoelder-Pichler-Tempsky, 1891, p. 117.

26. Hirschfeld, Isador. Interdental canals. J.A.D.A. 14:617 (April) 1927.

epithelial proliferation in other areas of the inflamed gingiva. The epithelium proliferates between and around the bundles of collagenous fibers and destroys the fibers by some desmolytic enzyme.[27, 28] The latter interpretation is strengthened by the observation of the active role of the epithelium during the eruption of continuously growing teeth.[29]

Histopathologically, marginal periodontitis can be characterized as chronic inflammation. The inflammatory cells are lymphocytes, plasma cells and macrophages. The plasma cells play an especially prominent role.

When the inflammation progresses into the most superficial marrow space of the septum, the bone at the crest of interdental septum is removed by osteoclastic resorption, widening the portal of entrance into the marrow spaces. This loss of the compact layer at the crest of the septum is the first roentgenologic sign of marginal periodontitis. The fatty marrow in the invaded marrow spaces is at the same time replaced by fibrous marrow and soon osteoclasts differentiate in contact with the bone. The osteoclastic resorption of the bordering bone finally allows the inflammation to break through into the periodontal membrane.

On the labial or lingual surface of the alveolar process the inflammation of the attached gingiva is accompanied by osteoclastic resorption of the alveolar plate. If this plate consists of a thin layer of compact bone, the inflammation soon reaches the periodontal membrane itself. Where the alveolar plate contains marrow spaces, as in the more basal parts of the alveolar process, the progress of inflammation toward the periodontal membrane occurs after involvement of the marrow spaces.

The inflammation reaches the periodontal membrane, according to these observations, after destruction of the alveolar bone proper, and at the same time detaches the principal fibers in the involved region. A periodontal membrane in the strict sense of the word has thus ceased to exist. Since no pressure arises in the involved part of the periodontal membrane, root resorptions are only rarely observed as a consequence of marginal periodontitis.[30]

Without therapy the progress of a marginal periodontitis will not cease. It leads, of course, to a progressive loosening of the teeth in consequence of the loss of bone and bony attachment.

Though, as mentioned before, marginal periodontitis is a chronic process, an acute exacerbation manifests itself as a lateral abscess when the drainage through the pocket is obstructed.

SYMPTOMATOLOGY

The clinical symptoms of marginal periodontitis can be understood easily from the pathogenesis and pathology.

The most important symptom that indicates the progress of the gingivitis into the deeper periodontal tissue, and thus justifies the diagnosis of marginal periodontitis, is loss of alveolar bone. The first sign of destruction of bone often is visible in the roentgenogram as a loss of the smooth outline of the crest of the interdental septum caused by the resorption of the most superficial compact plate. The considerable depth of the pocket that now can be termed a periodontal pocket is also characteristic of marginal periodontitis. Finally, there is the obvious sign of the loosening of the involved teeth.

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28. Aisenberg, M. S., and Aisenberg, A. D. Hyaluronidase in periodontal disease. Oral Surg., Oral Med. & Oral Path. 4:317 (March) 1951.

29. Wassermann, F. Personal communication.

30. Henry, J. L., and Weinmann, J. P. The pattern of resorption and repair of human cementum. J.A.D.A. 42:270 (March) 1951.

Periodontosis: a critical evaluation

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Periodontosis is a general term which refers to a "degenerative non-inflammatory destruction of the periodontium originating in one or more of the periodontal structures, characterized by migration and loosening of the teeth in the presence or absence of secondary epithelial proliferation and pocket formation or secondary gingival disease."[1] It is a chronic condition and, if permitted to run its course, ultimately results in the destruction of the periodontal tissues and loss of the teeth.

CLINICAL FEATURES

Periodontosis[2,3] affects both males and females and is seen more frequently in the period from puberty through the third decade. In the younger age groups it occurs more frequently in females.

The periodontal tissues of all the teeth may be involved or the condition may be limited to a single tooth or group of teeth. The incisor and first molar areas are the site of earliest and severest destruction. There is the least destruction of bone in the area of the mandibular bicuspids.

The clinical features of periodontosis vary according to the state of the disease.[4,5,6] Clinically the development and progress of periodontosis may be differentiated into a primary or degenerative stage and a secondary or inflammatory stage. In the early stage, tooth migration and mobility in the absence of notable gingival inflammation or pocket formation are characteristic. The usual picture consists of labiodistal migration and loosening of the maxillary incisors with diastema formation. Distal migration of the mandibular incisors is seen less frequently. The rate of migration is affected by the functional relations of the anterior teeth and pressure from the tongue. Elongation of the teeth is commonly associated with migration and loosening. Although it is generally limited to single rooted teeth, migration of multirooted teeth in either a mesial or distal direction is occasionally seen.

The secondary or inflammatory phase of the disease is characterized by the appearance of gingival inflammation, pocket formation and exudation. The pockets tend to be on the side of the tooth away from which the tooth is migrating. The severity of the changes that occur in the latter phase of the disease varies with the nature and distribution of local irritating factors, such as calcu-

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1. Report from the 1949 Nomenclature Committee of the American Academy of Periodontology. J. Periodont. 21:40 (Jan.) 1950.

2. Tenenbaum, B., and Karshan, M. Blood studies in periodontoclasia. J.A.D.A. 32:1372 (Nov.-Dec.) 1945.

3. Karshan, M., and others. Blood studies in periodontoclasia II. J. D. Res. 25:247 (Aug.) 1946.

4. Orban, B., and Weinmann, J. P. Diffuse atrophy of the alveolar bone (periodontosis). J. Periodont. 13:31 (Jan.) 1942.

5. Thoma, K. H., and Goldman, H. M. Classification and histopathology of paradontal disease. J.A.D.A. 24:1915 (Dec.) 1937.

6. Tenenbaum, B., and others. Clinical and microscopic study of the gingivae in periodontosis. J.A.D.A. 40:302 (March) 1950.

lus, food accumulation, food impaction and occlusal trauma. Lack of contact of the teeth is an additional unfavorable local environmental factor.

Periodontosis is characterized by a combination of the following roentgenographic features: (1) an arc-like contour of bone loss involving the first molar, which extends from the mesial surface of the second molar to the distal surface of the second bicuspid, coupled with bone loss in the incisor area, (2) thickening of the periodontal space, (3) absence or haziness of the lamina dura, (4) destruction of the interdental septa of the alveolar bone in a vertical rather than a horizontal direction, (5) a generalized alteration in the trabecular pattern of the alveolar bone, consisting of less clearly defined trabecular markings and increase in the size of the cancellous spaces, and (6) generalized loss of alveolar bone, which is accentuated in the incisor and first molar regions and least pronounced in relation to the mandibular bicuspids.

Periodontosis is basically a painless condition. However, the secondary stage of the disease may be accompanied by such symptoms as (1) sensitivity to thermal changes, foods or tactile stimulation, which results from denudation of the root surfaces, (2) dull deep radiating pain on chewing, which results from irritation of the periodontal tissues by the function of mobile teeth and the forceful wedging of food, (3) acute inflammatory symptoms, such as throbbing pain and sensitivity to percussion from periodontal abscess formation, and (4) pulpal symptoms, such as sensitivity to sweet, thermal changes or throbbing pain, which result from pulpitis associated with carious destruction of the root surfaces.

In periodontosis, the periodontal pockets are described as being usually of the infrabony type. This is an interesting observation; however, it does not imply that the presence of this type of pocket is evidence of the existence of periodontosis. In a histopathologic study, Carranza and Erausquin[7] reported that 25 per cent of the periodontal pockets examined were of the infrabony type. In addition, they noted that microscopically the type of bone destruction associated with the infrabony pocket is the same as that associated with other pockets; occlusal trauma is a common causative factor and the pocket is generally deeper on the pressure side of a tooth or the side toward which a tooth is inclined.

The term "infrabony" refers only to a morphologic variant of the periodontal pocket. It describes the relationship of the soft tissues of the pocket to the alveolar bone, which is pertinent insofar as it necessitates modification in treatment technics. The term "infrabony pocket" does not, however, indicate the existence of a specific periodontal disease with pathognomonic changes that differentiate it from other pockets.

ETIOLOGY

The exact cause of periodontosis has not been established. It is the consensus that the pathologic changes are systemic in origin. Trauma and inflammation are ruled out as causative factors. The causative systemic disturbances are referred to in such general terms as metabolic imbalance, hormonal disturbances, debilitating disease and nutritional deficiency. More specific reference to causative systemic disturbances include diabetes, syphilis, hypertension or collagen disease. A noninflammatory "betel periodontosis" has been described as a disease originating in the alveolar bone. The abrasive action of inveterate betel nut chewing[8] produces attrition and migration of the teeth, chiefly the maxillary incisors. Spreading, loosening and elongation of the maxillary incisors results from con-

7. Carranza, F. A., and Erausquin, R. Our first periodontal findings. Rev. Odontologica 27:485, 1939.

tinued impact of the mandibular incisors. The molar teeth show signs of attrition but are not otherwise affected.

The predilection of certain sites for periodontosis is attributed to local functional disturbances. Orban[9] noted that many different systemic conditions, rather than a single systemic disease, may be contributory. However, the systemic factor is not solely responsible. A local factor related to disturbance in the physiologic continuous eruption and normal migration of the teeth plus an inherited inferiority of the dental organ may be factors in the development of periodontosis.

NATURE OF THE UNDERLYING PATHOLOGIC CHANGES IN PERIODONTOSIS

There is a reasonable degree of agreement; first, regarding the clinical features of periodontosis and, second, regarding the fact that the condition described in recent years as periodontosis is the same as that described as "diffuse alveolar atrophy" by Gottlieb[10] in 1920. However, an attempt to determine the exact nature of the histologic changes responsible for the clinical findings in periodontosis meets with a somewhat less unified expression of opinion. In 1923, Gottlieb[11] stated the belief, based on microscopic study of two human jaws, that a deficiency in the continuous apposition of cementum initiated pathologic changes in diffuse alveolar atrophy. He later suggested that "deep cementopathia" might be a more appropriate term for this condition. Orban and Weinmann[4] divide periodontosis into the following correlated histopathologic and clinical stages:

The first stage is characterized by degeneration and desmolysis of the principal fibers of the periodontal membrane. This probably leads to cessation of cementum formation. There is simultaneous resorption of the alveolar bone due to (*a*) lack of functional stimulation from the tooth and (*b*) increased tissue pressure caused by edema and capillary proliferation. The increase in tissue pressure causes tooth migration. Clinically, tooth migration is the earliest sign of periodontosis and it occurs without any detectable inflammatory involvement.

The second stage is characterized by the rapid proliferation and migration of the epithelial attachment along the root. Proliferation of epithelial remnants in the periodontal membrane also may occur. The first signs of inflammatory involvement are seen in this stage. The inflammation is attributed to the penetration of toxins from the gingival sulcus which follow the course of the capillaries and epithelial structures. Clinically, both the first and second stages are of short duration and cannot be differentiated from each other. However, the first stage may be completely repaired if the cause is removed, while once the second stage has occurred, the third stage must follow.

The third stage is characterized by the development of deep periodontal pockets as the result of the separation of the proliferated epithelial attachment from the root surface. Inflammatory involvement of the periodontal tissues is aggravated because of irritation and infection from these deep pockets. Clinically, this is the stage that is observed most frequently.

Of particular interest is the finding that the initial degenerative change in periodontosis may affect the periodontal membrane of all the teeth or may be limited to a single tooth or even a localized area of a tooth.

In 1937 Thoma and Goldman[5] referred to periodontosis (parodontosis is

8. Balendra, W. The effect of betel chewing on the dental and oral tissues and its possible relationship to buccal carcinoma. Symposium on oral diseases in tropical countries. Brit. D. J. 87:83 (Aug. 19) 1949.

9. Orban, B. Classification of periodontal diseases. Paradentologie 3:159 (Dec.) 1949.

10. Gottlieb, B. Etiology and therapy of alveolar pyorrhea. Ztschr. f. Stomatol. 18:59, 1920.

11. Gottlieb, B. The new concept of periodontoclasia. J. Periodont. 17:7 (Jan.) 1946.

the terminology in the report) as follows: "It is of systemic origin and the bone is affected first. Parodontosis is therefore a disease of the alveolar bone in which the bone is transformed into fibrous tissue by reversion." According to these authors the initial lesion in periodontosis is in the alveolar bone. The bone is altered by the effect of poor nutrition or toxic processes on the bone cells, so that it undergoes disintegration when called on to bear the forces of function. In 1947 Goldman[12] described a condition similar to human periodontosis in two spider monkeys. The initiation and progress of periodontosis were described as occurring in three stages, comparable to those of Orban and Weinmann.[4] Microscopically the periodontal membrane showed the main pathologic changes. These consisted of (1) early dissociation of principal periodontal fibers with loss of functional structure, (2) break in the continuity of the periodontal fibers caused by noninflammatory degeneration arising in the middle of the fiber rather than at the cementum or bone and (3) loosening and swelling of the interfibrillar substance and disintegration of the periodontal fibers, with the formation of a loose, edematous connective tissue. Increased capillarity and edema were always present. In some instances only a portion of the periodontal membrane was involved; in others the entire periodontal membrane presented degenerative changes. Pocket formation occurred only when marginal degeneration of the periodontal membrane was present.

Resorptive changes in the alveolar bone were secondary to the changes in the periodontal membrane and resulted from local factors, such as pressure atrophy, loss of functional stimulation or inflammation arising from the presence of periodontal pockets, as distinct from the systemic origin of the primary changes in the periodontal membrane. Periodontosis was described as being not primarily a bone disease, because newly formed bone could be seen in the sockets of extruded teeth.

In 1949, Goldman[13] reported: "Microscopic studies show that periodontosis is essentially a disease of the periodontal membrane and alveolar bone that later affects the gingivae," and "changes in the periodontal membrane and alveolar bone are the earliest pathologic manifestations in periodontosis and are peculiar to the disease."

Among the changes described by different investigators in periodontosis is replacement of the normal fatty marrow of the alveolar bone about the involved teeth by a loose fibrous vascular connective tissue. The destruction of alveolar bone is described as "lacunar," involving the activity of osteoclasts, and "osteolytic" which is interpreted as destruction of the bone matrix and liberation of the osteocytes with resultant loss of bone substance.

Under the heading "periodontitis complex" Box[14] described a "systemic type" of periodontal disease resembling Gottlieb's "diffuse alveolar atrophy." Microscopically periodontitis complex is characterized by the early appearance in the periodontal membrane of an inflammatory tissue change induced by occlusal trauma, which is termed "rarefying pericementitis fibroma."

In a report of an animal experiment in which the acid-base balance was disturbed by the use of an excessively alkaline diet, Simonton[15] described the production of a destructive periodontal condition which he claimed resembled Gottlieb's diffuse alveolar atrophy. This condition was characterized by halisteresis, osteoporosis and lacunar resorption of

12. Goldman, Henry M. Periodontosis in the spider monkey—a preliminary report. J. Periodont. 18:34 (Jan.) 1947.

13. Goldman, H. M. Periodontia, ed. 2. St. Louis, C. V. Mosby Company, 1949, p. 130.

14. Box, H. Studies in periodontal pathology. Bulletin No. 7. Toronto, Canadian Dental Research Foundation, May, 1924.

15. Simonton, F. V. The etiology of parodontoclasia. Trans. 7th Int. Dent. Cong., Vol. 2. 1646, 1926.

alveolar bone. According to Simonton, periodontosis is a condition characterized by a retrograde metamorphosis of the alveolar bone in which inflammation, if present, is secondary. He suggested "parodontoclasia obscura" as an appropriate term for the condition.

Becks and Fleming[16] described "osteodystrophic type" of periodontosis in experimental animals on diets low in calcium and vitamin D. It is a condition characterized microscopically by primary osteoporotic or dystrophic changes in the alveolar bone which are duplicated in other areas of the skeleton, resorption of root surfaces and frequently generalized root end resorption. According to these investigators the osteodystrophic type of periodontosis is an outstanding type of periodontosis in man, occurring more frequently than is generally realized. It is not identical with Gottlieb's diffuse alveolar atrophy.

According to Tenenbaum[6] and others the microscopic picture of the gingivae in periodontosis changes as the condition progresses. In the early stages no pathologic changes are seen microscopically. In the later stages the gingivae present pronounced inflammatory infiltration and ulceration associated with periodontal pocket formation. There are, however, no characteristic microscopic changes in the gingiva whereby the condition can be identified as a specific disease entity.

In an effort to explore the problem of chronic destructive periodontal disease, particularly from the viewpoint of the basic nature of the effect of systemic influences, a series of animal experiments was undertaken by my co-workers and me in 1938. Within the limitations which govern the applicability of the results of animal experimentation to disease as it occurs in humans, the implications of the observations as they pertain to periodontosis will be discussed.

The initial experiment[17] dealt with the effect of acute starvation on the response of the periodontal tissues to artificially induced inflammation. We found, first, that bone loss may occur in periodontal disease as a localized feature of a generalized systemically induced skeletal disturbance and, second, that a generalized skeletal disturbance of systemic origin would increase the severity of alveolar bone loss associated with a local condition such as gingival inflammation.

Another opportunity to evaluate the effect of systemic disturbances on the periodontal structures presented itself with the introduction of the drug alloxan for the production of experimental diabetes mellitus.[18] Examination of the periodontal structures in experimental diabetes revealed a tendency toward varying degrees of nonspecific osteoporosis of alveolar bone unrelated to gingival changes in approximately 40 per cent of the diabetic animals. These animals also presented a reduction in the distribution and the amount of bone formation, an increase in bone resorption and a loss of bone substance associated with peripheral fragmentation and dissolution of alveolar bone matrix. All the bone changes occurred in the absence of gingival inflammation.

The loss of tooth-supporting bone in experimental diabetes appeared as a local manifestation of a generalized tendency toward osteoporosis, which affected not only the jaws but also the remainder of the skeletal system. The findings reemphasized the possibility that in periodontal disease neither the incidence nor the severity of alveolar bone destruction need be related to gingival changes.

The study of experimental diabetes was followed by investigation of the effect

16. Becks, H., and Fleming, W. C. Is osteodystrophic type of paradentosis, as produced in animals, identical with one form of paradentosis in man? (Abst.) J. D. Res. 15:206 (June-Aug.) 1935.

17. Glickman, I.; Morse, A., and Robinson, L. The systemic influence upon bone in periodontoclasia. J.A.D.A. 31:1435 (Nov.) 1944.

18. Glickman, I. The periodontal structures in experimental diabetes, New York J. Den. 16:226 (June-July) 1946.

of acute vitamin C deficiency on the periodontal tissues of the guinea pig.[19] It was observed that in acute vitamin C deficiency, the periodontal membrane presented edema, hemorrhage and pronounced collagen degeneration. The vessel channels and medullary spaces were enlarged and eroded. There was no new bone formation or osteoblastic activity in relation to the tooth-supporting bone. The over-all picture was one of generalized destruction of the periodontal tissues in the absence of any notable gingival inflammation or pocket formation.

The changes in acute vitamin C deficiency were comparable to those observed in experimental diabetes, in that there was loss of tooth-supporting bone unrelated to gingival inflammation or pocket formation. The changes observed in the vitamin C experiments, however, differed from those in diabetes in the nature of the bony changes and in the fact that, whereas both the periodontal membrane and alveolar bone showed severe destructive changes in vitamin C deficiency, only bone changes had been observed in experimental diabetes. Comparison of the findings in the two experiments is of interest because it demonstrates, first, that more than one type of systemic change may produce generalized periodontal destruction; second, that, although generalized loss of tooth-supporting bone may be a symptom common to different systemic disturbances, the detailed nature of the bone changes need not be the same; and, third, when a generalized loss of tooth-supporting bone occurs as the result of a systemic disturbance, the bone is not necessarily the only tissue involved, nor need it be the tissue in which the destructive changes are originally manifested.

After the nature of the periodontal changes arising from acute vitamin C deficiency was established, the next problem was to determine its effect, if any, on the response of alveolar bone to gingival inflammation. Gingival inflammation was artificially induced in vitamin C deficient and control animals.[20] As a result, the bone margin beneath the inflamed gingiva in the control animals presented both osteoclastic destruction and osteoblastic activity with new bone formation. Comparable areas in the vitamin C deficient animals, however, presented no new bone formation. Instead there was degeneration of a collagenous prebony matrix and osteoporosis of previously formed bone. Osteoblasts were rare. A prominent accumulation of connective tissue cells which separated the bony margin from the gingival inflammation and apparently gave rise to the osteoblasts in the control animals was absent in the vitamin C deficient animals. This retardation in the differentiation of connective tissue cells and ability to form and maintain new bone matrix accounted for the absence of well-formed new bone beneath the artificially induced gingival inflammation in the vitamin C deficient animals.

The vitamin C study added evidence in support of the existence of systemic regulatory influence on bone loss in destructive periodontal disease, which had been suggested by the previous findings. In addition, the vitamin C study demonstrated the specific manner in which the deficiency affected the response of alveolar bone to gingival inflammation. It was noted that, although acute vitamin C deficiency of itself resulted in loss of alveolar bone, artificially induced gingival inflammation was an added destructive influence. The bone loss arising from gingival inflammation was severer in the vitamin C deficient animals because of

19. Glickman, I. Acute vitamin C deficiency and periodontal disease. I. The periodontal tissues of the guinea pig in acute vitamin C deficiency. J.D. Res. 27:9 (Feb.) 1948.

20. Glickman, I. Acute vitamin C deficiency and periodontal disease. II. The effect of acute vitamin C deficiency upon the response of the periodontal tissues of the guinea pig to artificially induced inflammation. J.D. Res. 27:201 (April) 1948.

an inability on the part of these animals to maintain the existing alveolar bone or form new bone to replace that destroyed by the inflammation.

In an experiment conducted in the albino rat to evaluate the changes in the periodontal tissues associated with protein deprivation and to compare these changes with those observed in other areas of the skeletal system, Chawla and Glickman[21] reported the following observations:

1. In the periodontal area, degeneration of connective tissue of the gingiva and periodontal membrane, osteoporosis of the alveolar bone and retardation in the deposition of the cementum.

2. In the femur and vertebrae, osteoporosis, pronounced inhibition of the endochrondal and periosteal bone formation.

3. The changes observed in the periodontal membrane and alveolar bone were identical with those observed in the periosteum and bone in other areas of the skeletal system.

4. Osteoporosis in protein deprivation results from the following changes: reduced deposition of the osteoid, reduction in number of osteoblasts and retardation in the morphodifferentiation of connective tissue cells to form osteoblasts.

The observations in this study are of interest in that they reveal loss of alveolar bone which is primarily the result of the inhibition of normal formative changes rather than the introduction of destructive changes.

More recently,[22] in animals subjected to daily injection with 0.5 mg. of cortisone[23] for periods up to 43 days, miscroscopic examination of the periodontal structures revealed reduction in the height of alveolar bone, osteoporosis of alveolar bone characterized by pronounced reduction in the number of osteoblasts, the formation of new osteoid matrix, and a fibrinoid appearance of the periodontal membrane with a reduction in the number and degeneration of the collagen fibers of the periodontal membrane and gingival connective tissue. These changes occurred in the absence of gingival inflammation, inflammation being present only in isolated areas associated with local irritation. The changes in the alveolar bone and periodontal membrane were duplicated in the periosteum and bone areas of the skeletal system.

Comparison of the findings in this study with those in studies on protein deprivation reveals that loss of alveolar bone may represent similar microscopic features as the result of more than one type of systemic disturbance.

COMMENT

The theory that prompts persistent effort to isolate a "systemic type" of periodontal disease is presumptive. While it seeks for the mechanism of systemic participation in one type of case, it automatically assumes it is entirely absent in others. There is systemic participation in all cases of periodontal disease. It is herein demonstrated that the severity of bone loss that occurs where isolated zones of gingival inflammation are present is subject to systemic regulation. Cases of periodontal disease differ from one another, not in the complete absence or presence of systemic influence but rather in the degree of systemic participation and the relatively favorable or unfavorable nature of its effect.

The demonstration in a few animals or isolated autopsies that destructive

21. Chawla, T. N., and Glickman, I. Protein deprivation and the periodontal structures of the albino rat. Oral Surg., Oral Med. & Oral Path. 4:578 (May) 1951.

22. Glickman, I.; Stone, I. C., and Chawla, T. N. The effect of cortisone acetate (11 dehydro-17 hydroxycorticosterone-21 acetate) on the periodontal tissues of white mice. (Abst.) J.D. Res. 30:461 (Aug.) 1951.

23. A brand of cortisone acetate by Merck and Company, Inc.

periodontal disease may be initiated as noninflammatory degenerative changes in the periodontal membrane constitutes a valuable contribution in the periodontal field. Such findings do not justify the assumption that periodontosis is a specific disease that always originates in the same tissue. The experimental findings discussed herein indicate that systemically induced periodontal disease may originate in one or more of the periodontal tissues, depending on the nature of the causative factors. It is as fallacious to assume that all periodontal destruction originates in the same tissue as it is to postulate that there is only a single condition, local or systemic, capable of inducing periodontal disease.

The concept is untenable that there is a systemic type of periodontal disease that invariably presents certain clinical features without which a diagnosis of periodontosis cannot be made. Its attractiveness lies in its simplicity, which makes it expedient in clinical diagnosis. The possible variants in the clinical features of periodontal disease, primarily of systemic origin, are multiple, depending on the following factors: (1) the nature and severity of the systemic disturbance, (2) the stage of development of the jaws at which it first appeared and (3) local oral factors, such as the occlusal functional pattern and gingival inflammation.

Local factors are important in determining the clinical features of periodontal disease. The fact that a large percentage of patients with systemically motivated periodontal disease appear to present somewhat comparable clinical features is an indication that comparable local environmental factors are common to a comparatively large proportion of the human race. It does not indicate that the destructive changes in the periodontal tissues are being produced by a specific type of pathologic change.

That more persons with such local factors do not exhibit early migration, loosening and wandering of teeth in the absence of gingival inflammation may be due to one of the following facts:

1. The condition of the periodontal tissues is such as to withstand the effect of the local factors.

2. Systemically induced changes are present, but they are not sufficiently severe to jeopardize the physiologic status of the teeth and supporting structures.

With respect to treatment, it is important that one not minimize the contribution of local influences in producing the ultimate clinical changes in periodontal destruction which is primarily the result of systemic influences. The nature of a case may be such that by itself, neither the local nor systemic influence is sufficiently severe to produce clinically significant destructive changes. Elimination of the local contribution to the destructive changes often effects a clinical control of the disease even though minimal destructive tendencies of systemic origin may still exist. The concept that a patient either does or does not have periodontosis—and if the latter is true, the prognosis is hopeless—is discouraged by a basic understanding of the factors contributing to periodontal disease.

The effect of systemic influences on the periodontal tissues cannot be expressed in absolute positive or negative values. Instead, systemic influences result in a gradation of subtle morphologic changes. When small differences are created, the changes lead progressively from the normal to pronounced tissue loss. Systemic influences that result in tissue loss do not always produce degenerative changes. Tissue loss may be produced simply by a reduction in the normal formative processes or a retardation in the development of cells required for the normal formative processes. Considerable additional study with the use of such methods as histochemical and radiochemical technics remain to be employed to further clarify the nature of changes in the periodontal tissues which have escaped detection by usual histologic methods.

In dealing with destructive periodontal disease which is primarily systemically motivated, we are dealing not with a disease of the periodontium but with the effect of disease on the periodontium. It is in the subtle nature of the systemic effect on which interpretation of clinical phenomenon should be predicated. Results of the studies described herein suggest that all periodontal disease occurs as the result of an interaction between local and systemic influences. Cases differ from one another in the nature and degree of these influences. All gradations in the proportion of local and systemic participation occur. Although there are conditions termed "periodontosis" or "diffuse alveolar atrophy" or "negative bone factor"[24] in which systemic participation is the major factor, and although in a large number there are comparable clinical features, these conditions cannot be segregated as specific types of disease on the basis of any existing evidence. Instead, they represent extreme variants of destructive processes common to all periodontal disease.

SUMMARY

Continued adherence to the clinical viewpoint which attempts to segregate a "systemic type" of periodontal disease is discouraged because it appears to be inspired by a concept that is inconsistent with the basic nature of the effect of systemic influences on the periodontal tissues.

Differences exist in the clinical manifestation of periodontal disease, but such differences represent variants in a composite of subtle morphologic changes seen microscopically which await the application of newer research technics for their further clarification.

It is suggested that the quest for the elusive "systemic type" of periodontal disease be supplanted by a more critical comprehensive approach to the study of the interrelation of local and systemic influences in periodontal disease as they are manifested in tissue changes.

24. Glickman, I. The experimental basis for the "bone factor" concept in periodontal disease. J. Periodont. 20:7 (Jan.) 1949.

Bacterial Control • No single discovery has affected the welfare of man more profoundly than the demonstrations by Pasteur and his associates regarding the cause and spread of disease. The role of individual microorganisms has been laboriously and assiduously worked out and on the results of these researches men have devised methods for the improvement of industries and the protection of man and other animals. The individual methods are varied and complex but the principles on which they are based are the same. *Henry B. Ward, Foreword to Disinfection and Sterilization, by Ernest C. McCulloch, p. 7.*

Traumatism and atrophy: etiology, pathology and symptomatology

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An unusually large portion of the knowledge about periodontology has been the result of unsubstantiated opinion and, therefore, could not endure a scientific and objective appraisal. Often it has been the procedure to accept without question the assertions of "authorities" in periodontology. As a result, from time to time there have appeared statements, aphorisms, pronouncements or dogmas for which their originators have supplied no rational evidence emanating from previous studies or investigations.

On the subject of the etiology, pathology and symptomatology of traumatism and atrophy, there exist many assertions that need to be subjected to critical analysis.

Several classifications of periodontal disease have been proposed. The classifications that are now considered adequate are the ones that are based on scientific methods.

In his classifications of periodontal pathoses, Orban[1] based his distinctions on the clinical picture, etiology and histopathology of the disease process. He listed the following groups:

1. Gingivitis and periodontitis (inflammation).
2. Periodontosis (degeneration).
3. Periodontal hypertrophy (proliferative).
4. Periodontal atrophy (atrophy or retrogression).
5. Periodontal traumatism (traumatism).

This presentation will deal with an appraisal of the present information about the last two groups, periodontal atrophy and traumatism.

PERIODONTAL ATROPHY

Periodontal atrophy may be defined as a wasting away of the supporting tissues of the teeth. It is a retrogressive change manifesting itself as a receding of the gingivae, resorption of the crest of the alveolar bone and exposure of portions of the tooth root.

Orban[1] lists the following types of periodontal atrophy: (1) senile, (2) presenile, (3) local trauma, (4) disuse, (5) postinflammatory and (6) idiopathic. Essentially these types are similar to the various forms of general atrophic changes which Boyd[2] has indicated are the result of disuse, toxins, pressure, lack of nourishment, age and interference with the nerve supply.

Senile Atrophy of the Periodontium • There is a normal physiologic tendency

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1. Orban, B. Classification and nomenclature of periodontal diseases. J. Periodont. 13:88 (July) 1942.
2. Boyd, William A. Textbook of pathology, ed. 5. Philadelphia, Lea & Febiger, 1947.

for the periodontal tissues to recede with age. Clinically, this senile or "gerontal"[3] atrophy is evidenced by an enlarged clinical crown, which consists of the anatomic crown of the tooth together with that portion of the exposed cementum of the root. In the uncomplicated forms of senile atrophy, there is neither inflammation of the periodontal tissues nor pathologic pocket formation. Although the recession is generalized, the gingivae are healthy and the teeth are firm. Uncomplicated cases are rare, however, because of the morphologic changes that the recession produces. There is an increase in the tendency toward interdental retention of food and debris, and gingival inflammation often occurs.

The retrogressive changes usually affect the entire alveolar bone uniformly, with an equal level of bone resorption around all of the teeth and throughout the circumference of each tooth. Some teeth may be affected more than others. The labial surfaces of cuspids and mandibular central incisors and the lingual surfaces of the maxillary first molars are particularly susceptible.

Attrition of the masticating surfaces of the teeth is compensated by apical and bifurcation cementogenesis, which is a slow and continuous process throughout life. Orban[4] has pointed out that this continuous cementum deposition is normal and essential for the continuous eruptive movements of the functioning tooth and the perpetual reorganization of the periodontal membrane necessitated by these movements.

In the periodontal membrane, modifications associated with aging include a gradual retardation of mitosis, a lessened capacity for cellular accretion, cellular atrophy, degeneration, increased cell pigmentation and fatty infiltration. The chemical nature of the cell may vary during aging without an accompanying morphologic change.[5] The nerve cells undergo true fatty degeneration. The blood vessels lose their elasticity and are hardened through deposition of fibrous tissue and calcium. The decreased blood flow impairs tissue nutrition and results in some accumulation of waste products. White connective tissue fibers are inadequately replaced, since the old fibers are not removed like cells, but tend to remain in the tissues.

In the alveolar bone, changes associated with aging include increased fragility, decreased vascularity, lowered metabolic rate, some degree of fibrosis of the bone marrow and a lessened ability to heal. The crests of the alveolar process resorb, and the trabeculae atrophy. Resorption of the alveolar crest is accompanied by gingival inflammation. If attrition of the masticating surfaces of the teeth parallels the alveolar changes of senile atrophy, periodontal disease may be avoided. Actually, the more conservative the rate of alveolar bone atrophy, the better the prognosis in the retention of teeth.

In senile atrophy, the epithelium of the gingivae proliferates apically until it is attached only to the cementum of the root.[6] This downward epithelial proliferation is accompanied by round cell infiltration and is associated with the afore-mentioned changes in the periodontal membrane and alveolar bone. The receded gingivae are more prone to injury and heal less readily.

In his discussion of changes in the oral tissues associated with aging, Thomas[7] pointed out the need for utilizing all fields of research and coordinating all phases of the basic sciences in order to understand the details of such changes in the oral tissues of humans.

3. Rothner, J. T. Clinical changes of the gingiva in periodontal diseases. J.A.D.A. 37:289 (Sept.) 1948.

4. Orban, B. Oral histology and embryology, ed. 2. St. Louis, C. V. Mosby Co., 1949.

5. Stieglitz, E. J. Pertinent problems of geriatric medicine. Ann. Int. Med. 18:89 (Jan.) 1943.

6. Gottlieb, B. Der Epithelansatz am Zahne. Deutsche Monatschr. f. Zahnh. 39:42 (March) 1921.

7. Thomas, B. O. A. Gerodontology: the study of changes in oral tissues associated with aging. J.A.D.A. 33:207 (Feb.) 1946.

Presenile Atrophy and Idiopathic Atrophy • Presenile atrophy has also been referred to as "precocious advanced alveolar atrophy,"[8] "paradentose juvenile,"[9] "juvenile atrophy"[10] and "atrophia praecox."[11] This condition is characterized by the occurrence of atrophic changes usually associated with age in the periodontal tissues of young healthy persons.

The cause of presenile atrophy has been termed "idiopathic," therefore it is convenient to discuss these conditions at the same time. The term "idiopathic" is applied when atrophy of the tissues of the periodontium occurs without any obvious cause. More especially is the term pertinent when gingival recession occurs at early ages and exposes extensive portions of the root. Zappler[12] expressed the belief that idiopathic alveolar atrophy is encountered with increasing frequency at puberty and adolescence. She reported a case of gingival atrophy on the labial surface of the upper anterior teeth in a 3 year old child. Weinmann and Orban[13] indicated that they had never seen periodontal atrophy in the deciduous dentition.

Clinically, the soft tissues in presenile atrophy appear anemic and glisten. There is no deepening of the gingival sulcus, no apparent inflammation in uninvolved cases, and no purulent discharge. The recession of the gingivae tends to be symmetrical and the cementum is exposed. Resorption of the alveolar crest and corresponding portion of the periodontal membrane are additional symptoms. As a matter of fact, the clinical and histologic manifestations of presenile atrophy are identical with those retrogressive periodontal changes previously described in physiologic senile atrophy.

Miller[14] described precocious advanced alveolar atrophy as a rapid pathologic passive eruption of the tooth. He noted that maxillary and mandibular incisors and first molars were the teeth most often involved, and on the basis of his observations he described three types of precocious advanced alveolar atrophy: (1) early (5 to 7 years of age, involving incisors and molars), (2) late (11 to 13 years of age, involving cuspids, bicuspids and second molars) and (3) combined (early involvement of all teeth).

A survey of the literature indicates that, although a great amount of investigation has been carried on in the past, the cause of presenile atrophy is still unknown. Some investigators[15] believe that in the more unusual cases of presenile alveolar atrophy, occurring in persons under 20 years, especially in females, an insufficiency of sex hormone from the gonads and adrenal cortex is responsible. Roy[10] correlated alveolar atrophy with the appearance of gonadal hormones. Talbot[16] and Kranz and Falik[17] presumed that evolutionary changes in the masticatory apparatus were responsible for presenile atrophy. Miller and Seidler[8] suggested that developmental aberrations and nutritional and metabolic diseases were causative. Hulin[9] ascribed the condition to inherited pluriglandular dysfunction. Vincent[18] pointed out that the stepped up nervous tension and excitement in children and the emotional and psychologic imbalances are factors not to be neglected in the study of causes of early retrogressive changes, since they do

8. Miller, S. G., and Seidler, B. B. Periodontoclasia in the young. New York J. Den. 10:423 (Nov.) 1940.

9. Hulin, C. Les paradontoses pyorrheiques. Paris, R. Foulon, 1941.

10. Roy, M. Les formes cliniques de la pyorrhée alvéolaire. L'Odontologie 71:433, 1933.

11. Merritt, A. H. Periodontal diseases, ed. 3. New York, Macmillan Co., 1945, p. 43.

12. Zappler, S. E. Periodontal disease in children. J.A.D.A. 37:333 (Sept.) 1948.

13. Weinmann, J. P., and Orban, B. Personal communication to S. E. Zappler.

14. Miller, S. C. Textbook of periodontia, ed. 3. Philadelphia, Blakiston Co., 1950, p. 663-4.

15. Kronfeld, R. Histopathology of the teeth and their surrounding structures, ed. 3. Philadelphia, Lea & Febiger, 1949.

16. Talbot, E. S. Interstitial gingivitis or so-called pyorrhea alveolaris. Philadelphia, S. S. White Dental Mfg. Co., 1899.

17. Kranz, P., and Falik, K. Alveolar pyorrhoe. Berlin, H. Meusser, 1922.

18. Vincent, Elizabeth L. Mental, social and emotional factors in adolescence. In Brennemenn's Practice of Pediatrics, vol. 1, Hagerstown, Md., W. F. Prior Co., 1948.

produce emotional maladjustments in adolescence that are reflected in physical disorders.

The evidence presented to substantiate these factors as being causative of presenile atrophy is, however, not conclusive. It appears, therefore, that in this phase of periodontology there is need for extensive research to determine the causes, selectivity in site, manner of occurrence and rapidity of onset of presenile atrophy.

Atrophy from Local Trauma and Postinflammatory Atrophy • Periodontal atrophy occurs as a sequence of local trauma and inflammation. Among the different forms of local trauma listed are incorrect toothbrushing, incorrect use of toothpicks and dental floss, calculus, food impaction, injurious dentifrices, improper restorations, and chemical and atmospheric irritants.

Miller[19] stated that alcohol and tobacco frequently are indictable chemical irritants and that "typical cases of alcoholic gingivitis with marginal wasting of the gingivae which used to be prevalent in the prohibition era are still appearing." Blankenhorn and Spies[20] reported the occurrence of reddened swollen gingivae in 73 alcoholics with pellagra. Recession of the gingival tissues was not recorded in their findings.

Hirschfeld[21] made a careful study of the effects of incorrect toothbrushing and divided the causative or predisposing factors of toothbrush trauma and recession into three groups: (1) dental arch abnormalities, (2) improper toothbrush technic and (3) faulty toothbrush. His conclusions were to the effect that incorrect toothbrushing can and does cause gingival recession through pressure atrophy. In these cases there are hard pink marginal crescents on the labial and buccal surfaces of the gingivae, while the lingual and palatal gingivae retain their normal color, form and position. The depth of the gingival sulcus and the color of the tissues remain normal, regardless of the extent of recession. In addition, there is no appreciable degree of tooth mobility beyond the normal, since alveolar resorption occurs only on that surface of the tooth which is affected by the atrophic process. The cuspid region is usually most affected, the process diminishing somewhat at the second bicuspid. The diagnosis of toothbrush trauma may be corroborated by the patient's demonstration of his incorrect toothbrushing.

Miller[19] expressed the opinion that rotary or cross brushing of the scrubbing type, especially when performed with highly abrasive dentifrices, produces horizontal gingival recession and tooth surface abrasion. Vertical brushing also is harmful, its prime effect being on the gingival tissue. It is generally agreed that, besides improper toothbrushing technic, overvigorous application of the brush, lack of an orderly procedure and forcing the bristles in the gingival sulcus are other important factors in the production of toothbrush abrasion and injury of the gingivae.

Hirshfeld[21] asserted that many of the Stillman clefts usually attributed to traumatic occlusion actually are caused by repeated injury from incorrect toothbrush usage.

Improper use of the toothbrush and dental floss may produce periodontal atrophy. The forceful mechanical thrust of food between the teeth and against the gingivae is termed "vertical food impaction."[22] It creates an irritant effect. Initial reactions to this irritation to soft tissues are inflammation, hemorrhage and hypersensitivity. These same tissue reac-

19. Miller, S. C. Textbook of periodontia, ed. 3. Philadelphia, Blakiston Co., 1950, p. 84, 85.

20. Blankenhorn, M. A., and Spies, T. D. Oral complication of chronic alcoholism. J.A.M.A. 107:641 (Aug. 29) 1936.

21. Hirschfeld, Isador. The toothbrush, its use and abuse. Brooklyn, N. Y., Dental Items of Interest Publishing Co., 1939.

22. Hirschfeld, Isador. Food impaction. J.A.D.A. 17: 1504 (Aug.) 1930.

tions occur from supragingival calculus and the various other forms of mechanical irritation. After the inflammatory processes subside, the gingivae shrink and the root surface is exposed. There is an apical shift of the epithelial attachment along the cementum of the tooth root.

In a discussion of pocket formation and gingival recession, Gottlieb[23] stated that, although both have in common the speeded apical movement, or recession, of the base of the sulcus, yet the gingival margin followed the base, thus causing the depth of the sulcus to remain the same. The apical movement of the base of the sulcus was not caused by calculus or inflammation. He suggested that these gingival recessions were the result of lack of cementum formation. The absence of an effective cementum barrier caused the epithelial attachment to grow downward. This was followed by gingival recession, simple pocket formation or formation of an intrabony pocket. Topographic conditions of the involved area determined the result. Gottlieb used the term "marginal cementopathia" to describe the pathologic condition of the cementum adjoining the epithelial attachment, which in turn was responsible for gingival recession and pocket formation.

Zappler[12] stated that postinflammatory atrophy is more commonly seen after puberty than prior to that time. Gingival recession may occur secondarily, following inflammatory processes. The gingivae shrink and portions of the root surface may become exposed after the inflammation subsides.

In acute necrotizing ulcerative gingivitis, the initial lesions occur in focal incubation zones, such as a deep gingival crevice. A decided reduction in oxygen tension results. Necrosis and sloughing of the gingivae follow and the punched-out appearance of the interdental papillae is produced.[24] This necrotizing process eliminates the sulcus and alters permanently the previous architectural form of the interdental papillae. Subsequent to the necrotizing processes, the disease becomes chronic. Ulceration continues to deform the interproximal region and concave contours are produced. This deformity may be regarded as a gingival recession and is generally conceded to be a form of postinflammatory atrophy.

Disuse Atrophy • This form of atrophy occurs in the periodontal tissues as a result of lack of function. Such lack of function is exemplified in nonocclusion, an abnormal condition in which one or more teeth cannot be brought into contact with their opponents regardless of the position in which the mandible is moved. Goldman[25] uses the term "functional insufficiency" to describe the form of atrophy and lowered tissue resistance resulting from lack of function. He differentiates between a total insufficiency of the jaws resulting from lack of adequate masticatory function, and a localized insufficiency of the supporting tissues around certain teeth without antagonists.

Changes in the width of the periodontal membrane during functional insufficiency were investigated by Preissecker,[26] who removed the molars on one side of the jaws of young albino rats. At the end of a six month period, the width of the periodontal membrane around the nonfunctioning teeth was approximately half that of the functioning teeth. Preissecker distinguished between the physiologic periodontal membrane width in nonfunctioning and that in functioning teeth.

Coolidge[27] measured the width of the periodontal membrane in 172 teeth from

23. Gottlieb, B. The new concept of periodontoclasia. J. Periodont. 17:7 (Jan.) 1946.

24. Lyons, Harry. Acute necrotizing periodontal disease: an appraisal of therapy and a critical criterion. J.A.D.A. 37:271 (Sept.) 1948.

25. Goldman, H. M. Periodontia, ed. 2. St. Louis, C. V. Mosby Co., 1949.

26. Preissecker, O. Influence of experimental disuse on the peridental membrane, (Abst.) J. D. Res. 11: 518 (June) 1931.

27. Coolidge, E. D. The thickness of the human periodontal membrane. J.A.D.A. 24:1260 (Aug.) 1937.

15 human jaws. He concluded that the average thickness of the periodontal membrane of those teeth subjected to heavy function was 0.18 mm. and in those teeth without antagonists the thickness was 0.13 mm. He concluded that the thickness of the periodontal membrane in each jaw was in direct proportion to the intensity of masticatory function.

Miller[28] listed the following causative factors in nonocclusion: (1) habits, (2) anomalies of development, (3) disease, (4) injury to oral structures and (5) faulty restorations.

Clinically, lack of function and lack of stimulation of the gingivae result in loss of tone of the latter. Recession of the gingivae is not a prominent symptom in disuse atrophy. Extrusion of the tooth occurs and there is an increase in thermal sensitivity. Gingival recession or deep pocket formation is seen when nonocclusion is due to the extraction of the opposing teeth with no replacement.

Histopathologically, the periodontal membrane becomes thinner and degenerates.[29] The principal fibers lose their regular arrangement and the periodontal membrane itself becomes a loose, irregularly arranged connective tissue.

The cementum becomes thicker than that of the functioning teeth in the same jaw. This hypertrophic cementum lacks Sharpey's fibers and usually is arranged evenly around the root surface. In bone, because of lack of tension and lack of stimulus to produce new bone, there is a reduction in the total bulk, in the form of localized osteoporosis. Trabeculae are fewer and thinner. The supporting bone is almost entirely missing, so that only fatty marrow is present. As is the case with cementum, there is a lack of Sharpey's fibers in the alveolar bone.

Weinmann and Sicher[30] point out that hypofunction leads to venous, capillary and arterial stasis, with engorgement, edema and swelling of the blood vessels. This increase in size of the blood vessels exerts pressure, which is transmitted to the bone. The increase in pressure is greater than the physiologic limit intended for bone and, as a consequence, resorption occurs.

Goldman[25] noted that the problem of alterations in alveolar architecture under various influences has as yet not been fully investigated.

PERIODONTAL TRAUMATISM

There is no aspect of periodontology that has received more attention than traumatic occlusion. The literature on the subject is voluminous, a great portion of it being devoted to argumentation about whether it is a primary or secondary cause of periodontal disease. At present, it appears to be the consensus that trauma is not the primary causative factor in periodontal disease, but rather an aggravating factor when some form of periodontal disease already exists.

"Traumatic occlusion" is a term introduced by Stillman[31] in 1917. "Traumatogenic occlusion,"[32] "occlusal trauma" or stress, "occlusal traumatosis,"[11] "pressure pericementitis,"[33] "occlusal overstress"[34] and "dynamic irritation"[35] are other terms that have been advanced by various clinicians from time to time and are more or less synonymous with traumatic occlusion.

28. Miller, S. C. Non-occlusion as a factor in periodontal disease. J. D. Res. 8:603 (Oct.) 1928.

29. Box, H. K. Studies in periodontal pathology. Bulletin No. 7. Toronto, Canadian Dental Research Foundation, May, 1924.

30. Weinmann, J., and Sicher, H. Bone and bones. St. Louis, C. V. Mosby Co., 1947.

31. Stillman, P. R. The management of pyorrhea. D. Cosmos 59:405 (April) 1917.

32. Box, H. K. Traumatic occlusion and traumatogenic occlusion. Oral Health 20:642, 1930.

33. Black, G. V. Operative dentistry, vol. 4, ed. 7. Chicago, Medico-Dental Publishing Co., 1936.

34. Rule, R. W.; Wright, R., and Smith, T. S. A symposium on the etiology, diagnosis and treatment of periodontal disease. J. California D. A. 18:117 (July-Aug.) 1942; 18:151 (Sept.-Oct.) 1942; 18:192 (Nov.-Dec.) 1942; 19:2 (Jan.-Feb.) 1943; 19:33 (Mar.-April) 1943.

35. Gretzinger, Max. Dynamic irritation as a cause of periodontal disease and the means for its elimination. J.A.D.A. 37:294 (Sept.) 1948.

"Traumatic occlusion" is defined as an abnormal occlusal stress which is capable of producing or has produced an injury to the periodontium. Although there are a significant number of persons in whom the overloading of the teeth for long periods has produced no visible damage to the periodontium, unusual stresses on the teeth do produce damage to both the teeth and the supporting structures in a large number of cases. Experimental studies[36-40] and histopathologic studies of sections of human jaws at autopsy have proved that occlusal trauma is a definite cause of certain pathologic disturbances in the periodontal membrane. The observations in human autopsy material which substantiate the presence of occlusal trauma include abrasion, cemental tears, root and alveolar bone resorption, periodontal membrane injuries and fracture of the teeth. The first comprehensive study of the role of occlusal stress on teeth in relation to periodontal disease was made by Talbot,[16] who pointed out that man is predisposed to disease of the supporting tissues of the teeth because jaw function has been greatly decreased as a result of modern methods of food preparation. Studies of skulls of primitive and modern man confirm the observation that powerful occlusal function leads to well-developed jaws and teeth, good alignment and freedom from disease, while slight function produces weak maxillae and mandibles.

Stillman[41] is credited with being the first to emphasize traumatic occlusion as a cause of periodontal disease. Repeated abnormal pressures of one tooth on another produce traumatic injury. He pointed out that there are noninfectious changes that are directly produced by traumatic occlusion. In time, as a result of continued irritation, a chronic suppurative infection usually occurs. McCall[41] supported Stillman in the belief that, once traumatic occlusion is established, irritation and periodontal congestion result.

In studies of human teeth, Orban and Weinmann[42] found signs of trauma in every case of missing teeth. Gottlieb and Orban[43] studied the effects of traumatic occlusion in dogs. They found that the periodontal tissue in the area of pressure became thinner and bone resorption enlarged the periodontal space. On the opposite side, tension stimulated bone building.

Box[32] experimented on sheep to observe the effects of excessive occlusal stress and found that continued excessive stress tended to move teeth out of line. He differentiated between traumatic and traumatogenic occlusions, pointing out that the latter represented "an occlusion which, under biting pressure was capable of producing or has produced injury to the periodontal tissues."

Breitner[44] pointed out that histologic studies have shown that natural abrasion of the teeth is a protective factor against damage to the tooth-supporting apparatus, since it reduces forces acting laterally on the teeth. He said that purely lateral forces can be tolerated only to a limited extent, since only a small number of principal fibers of the periodontal membrane are designed to resist lateral forces. Vertical forces are easily assimilated. According to Breitner, the intraoral

36. Gottlieb, B., and Orban, B. Experimental traumatic occlusion. Leipzig, G. Thieme, 1931.

37. Gottlieb, B., and Orban, B. Tissue changes in experimental traumatic occlusion, with special reference to age and constitution. (Abst.) J. D. Res. 11:505 (June) 1931.

38. Schwarz, R. Tissue changes incident to orthodontic tooth movement. Internat. J. Orthodontia 18:331, 1942.

39. Stuteville, O. H. Injuries to the teeth and supporting structures caused by various orthodontic appliances, and methods of preventing these injuries. J.A.D.A. 24:1494 (Sept.) 1937.

40. Oppenheim, A. Tissue changes, particularly of the bone, incident to tooth movement. Am. Orthodont. 3:57, 1911 3:113, 1912.

41. Stillman, P. R., and McCall, J. O. Textbook of clinical periodontia. New York, Macmillan Co., 1922.

42. Orban, B., and Weinmann, J. Signs of traumatic occlusion in average human jaws. J. D. Res. 13:216 (June) 1933.

43. Gottlieb, B., and Orban, B. Biology and pathology of the tooth and its supporting mechanism. New York, Macmillan Co., 1938, p. 58-105.

44. Breitner, C. Tooth-supporting apparatus under occlusal changes. J. Periodont. 13:72 (July) 1942.

forces of the musculature of the lips and cheeks against the teeth and the tongue can and do produce damage to the periodontal tissues.

Leonard[45] stated that uneven wear of the teeth produces changes in the directions of forces that hold the teeth in equilibrium. As a result, interferences in the masticatory movements occur and positions of stability are impossible to achieve.

McCoy[46] stressed the importance of the presence of all teeth and the integrity of the form and relationship of each tooth in maintaining the harmonious action of cusps and crowns. Tipping of the teeth as a result of tooth loss is, therefore, a causative factor of occlusal trauma.

Gratzinger[35] expressed the belief that the damaging effects to the periodontal tissues do not have the character of a traumatic injury and are not produced by some definite type of occlusion. The damage is the effect of a force active for a long time. This chronic irritating force is lateral in direction. It is developed from the muscles of mastication, and a lack of balance between the retentive power of the cheek and lip muscles, and the force of the tongue muscles. Since the forces not related to occlusion constitute the source of irritation, Gratzinger prefers the term "dynamic irritation" to traumatic occlusion.

In a discussion of tissue changes associated with occlusal stresses, Lundquist[47] demonstrated that normal bony structures accompanied normal functional demand. There was an increase in the density of bone when functional demand increased and a decrease in density when functional demand was lessened. Increased functional demands might become excessive and cause injury to the periodontal tissues in persons who have experienced a diminution in vigorous reactive capacity. Such a traumatic injury should be considered an aseptic wound.

Glickman and Wood[48] found that bone resorption or bone building occurring adjacent to the periodontal membrane was paralleled by similar changes in other parts of the jaw and skeletal system. Further studies with the endocrines and vitamins have shown that deleterious changes in the periodontal tissues do occur as a result of aberrations in activity of the endocrine glands and deficiencies in utilization of vitamins. Boyle and Bessey[49] showed that periodontal disease may develop at an early age in animals deficient in vitamin A, which plays a role in the cornification of epithelial tissues. Moreover, the alveolar bone then showed evidence of failure to withstand functional stress. Boyle concluded that inadequate diet, poor absorption of food, vitamins and minerals, and disturbed metabolic activity were conditions that handicap body tissues in maintaining normal function.

The controversial status of the occlusal factor and its major or minor etiologic relationship to periodontal disease is revealed in a survey of the literature. MacMillan[50] is in definite disagreement with the opinion that traumatic occlusion is the predominating causative factor in periodontal disease. In stating his case, he pointed out a number of discrepancies in the doctrine of traumatic occlusion. Among these was the fact that the maximum amount of stress which the periodontium is able to withstand innocuously has not been determined. Nor has there been a determination of the critical point at which normal stimulation ends and

45. Leonard, H. J. The occlusal factor in periodontal disease. J. Periodont. 14:12 (Jan.) 1943.

46. McCoy, J. Applied orthodontia. Philadelphia, Lea & Febiger, 1935.

47. Lundquist, G. R. Connective tissue changes associated with variable occlusal stresses. J.A.D.A. 24: 1577 (Oct.) 1937.

48. Glickman, I., and Wood, Harold. Bone histology in periodontal disease. J. D. Res. 21:35 (Feb.) 1942.

49. Boyle, P. E., and Bessey, O. A. The effect of acute vitamin A deficiency on the molar teeth and paradontal tissues, with a comment on deformed incisor teeth in this deficiency. (Abst.). J. D. Res. 20:236 (June) 1941.

50. MacMillan, H. W. The case against traumatic occlusion. J.A.D.A. 17:1996 (Nov.) 1930.

retrogressive changes as a result of masticatory overstress begin. Furthermore, the clinical practice of disoccluding teeth has neither prevented nor cured periodontal disease. He concludes that the degenerative process attributed to traumatic occlusion could not be produced on a perfectly healthy tooth by masticatory forces.

In a discussion of the physiologic processes of the periodontal membrane, Blayney[51] disagreed with the concept that too much pressure produces alveolar bone and cementum resorption. He expressed the belief that lack of function causes bone resorption and periodontal membrane loss on the side toward which the tooth is being thrust by occlusal forces. An intermittent pressure as produced by occlusal function would result in bone building rather than resorption, since the latter requires a constant pressure. This concept emphasized the superior influence of static over dynamic conditions in the matter of bone resorption.

The work of Leriche and Policard[52] has been accepted as representing a sound approach to the subject of bone physiology, so that it is reasonable to accept as factual their statement that constant pressure on bone produces resorption, while intermittent pressure favors bone formation.

Kronfeld[15] stated that in persons with high resistance, even a great amount of occlusal trauma does not produce resorption of the root, while in patients susceptible to root resorption, normal mastication occasionally may produce progressive root resorption.

Rule[34] insisted that occlusal overstress is not a primary factor in the initiation of periodontal disease. He considered it a secondary predisposing factor, operating almost exclusively in cases in which the foundational structures have become weakened through dystrophic or other predisposing factors.

Merritt[11] stated that trauma is a symptom of periodontal disease and not the cause of it. The breakdown in tissue resistance is not the result of an excessive occlusal force, but rather emanates from a normal force applied to tissues that for some reason have lost their power to resist it. He used the term "occlusal traumatosis" to designate the effect produced on tissue by traumatic occlusion.

Coolidge[53] has summarized the local causes of occlusal trauma as follows: (1) unrestored loss of posterior teeth with tooth drift, (2) malposition of the teeth, (3) cuspal interference, (4) uneven wear of the teeth, (5) atrophy of the supporting tissues of the teeth, (6) excessive overbite, (7) uneven eruption rate, (8) abrasion with loss of vertical dimension, (9) defective occlusion as induced by ill-constructed artificial restorations and orthodontic appliances and (10) injurious oral habits.

Goldman[26] pointed out that systemic factors may influence and complicate these local causes of traumatic occlusion. The many complex reactions occurring in the periodontium when both excessive stress and chronic inflammation are present make it difficult for one to evaluate the systemic relationship to periodontal disease. An established systemic disease does not presuppose a cause and effect periodontal relationship. The researches of competent investigators would tend to disqualify a simple cause and effect relationship between occlusal stresses and deterioration of the periodontium. Until more convincing evidence to the contrary is produced, Orban's[54] conclusion that the general physical state may be an important determinant of tissue changes in traumatic occlusion would seem to be the most logical.

51. Blayney, J. R. The physiology of the periodontal membrane. Illinois D. J. 8:64 (Feb.) 1939.

52. Leriche, R., and Policard, A. The normal and pathological physiology of bone. St. Louis, C. V. Mosby Co., 1928, p. 99.

53. Coolidge, E. D. Clinical pathology and treatment of dental pulp and periodontal tissues. Philadelphia, Lea & Febiger, 1939.

54. Orban, B. Traumatic occlusion and gum inflammation. J. Periodont. 10:39 (Jan.) 1939.

The clinical picture of periodontal traumatism as a cause of periodontal disease has been described by Orban[1] as a loosening of teeth due to widening of the periodontal space. There is a sensitiveness to percussion and thermal change. If trauma is in a single direction, the tooth may move out of occlusion. Box[52] and Leonard[45] have recorded several other symptoms of periodontal disease resulting from traumatic occlusion. These include tenderness to biting stress, traumatic crescents, congestion of the marginal gingivae, McCall's festoons (marginal thickening of the gingiva), Stillman's clefts (slits in the gingival margin over a pocket) and changes in tooth stability. Roentgenographically there are thickenings of the lamina dura, rarefaction, condensation and hypercementosis in some cases and resorption of the root in others.

The pathologic changes occurring as a result of occlusal trauma have been summarized by Orban.[1] Trauma may act in vertical (axial) or horizontal (lateral) direction. Vertical overloading may not lead to degenerative changes, since the fiber arrangement of the periodontal membrane can and does compensate the overstress. Lateral or horizontal forces compress the periodontal membrane between the tooth root and the alveolar wall; if the forces are benign, a moderate degree of inflammation is produced. This causes the bone along the alveolar wall to resorb together with the principal fibers in that region. If the traumatic forces are severe and persistent and the tissue resistance is low, then thrombosis, hemorrhage and eventually necrosis of the periodontal tissues result. Extensive bone resorption occurs and the periodontal space becomes widened.

Orban[55] has demonstrated the presence of cartilage in the periodontal membrane in cases of occlusal trauma. When occlusal trauma is in a single direction, resorption of bone is found in the pressure zone and bone building takes place in the traction zone. When the trauma is in several directions, bone destruction occurs all around the root surface, because of recurring pressure. Widening of the periodontal membrane space and loosening of the teeth follow, and mastication is impaired.

Box[56] pointed out that injured portions are altered by proliferation of fibrillar connective tissue about the partially occluded blood vessels at the expense of normal white connective tissue, periodontal membrane fibers and bone. The greatest change occurs at the alveolar border, where movement is greatest. Gottlieb[57] showed that the resorption of the principal fibers adjacent to the epithelial attachment permits apical proliferation of the epithelium. This predisposes to a deepening of the gingival sulcus and mechanical tears tend to split the epithelium.

Resorption of the cementum occurs in many cases when teeth are subjected to excessive lateral stresses. It is not known why resorption takes place in certain cases and not in others, especially when external conditions are identical. Becks[58] expressed the opinion that systemic disorders are influential in extensive cementum resorption. Occlusal trauma of long standing is capable of causing cementum resorption, which is never as extensive as bone resorption and is usually repaired as soon as the trauma subsides. If teeth are involved in acute trauma, fragments of cementum may be torn from the dentin.

Opinions vary as to the gingival changes resulting from traumatic occlusion. Kronfeld[18] and Orban[55] agreed that, if tissue resistance is high, there are

55. Orban, B. Tissue changes in traumatic occlusion. J.A.D.A. 15:2090 (Nov.) 1928.

56. Box, H. K. Twelve periodontal studies. Toronto, Univ. Toronto Press, 1940.

57. Gottlieb, B. Traumatic occlusion. J.A.D.A. 14: 1276 (July) 1927.

58. Becks, Hermann. Systemic background of paradentosis. J.A.D.A. 28:1447 (Sept.) 1941.

not likely to be any pathologic changes in the gingivae resulting directly from traumatic occlusion. Thoma[59] stated that gingival changes do not, as a rule, occur as a direct result of abnormal stress, since increased function is more likely to influence the gingivae favorably. Hill,[60] however, listed occlusal tramatism as one of the important causes of gingivitis. He expressed the belief that circulatory changes induced in the periodontal tissues by excessive occlusal stress are reflexly conveyed to the gingiva.

SUMMARY AND CONCLUSION

There is a large amount of information available on periodontal traumatism and atrophy, as well as confusion and lack of conclusive experimental evidence to confirm prevalent opinions.

Many of the causative factors of traumatism and atrophy have not as yet been fully explained. The generally controversial state of pronouncements on the topic is indicative of a profound need for verification of the theories of the past and careful analysis of many of the studies of the present. For the future there appears to be an urgent demand for a systematic investigation of neglected areas. One of the most attractive avenues for exploration is in the field of psychosomatic medicine and dentistry. Psychosomatic dentistry may and probably does have some positive contributions to make to the subject of traumatism and atrophy. Like any other branch of dentistry, it is worthy of scientific research and clarification.

59. Thoma, K. H. Oral pathology, ed. 2. St. Louis, C. V. Mosby Co., 1944, p. 455.

60. Hill, T. J. Textbook of oral pathology, ed. 3. Philadelphia, Lea & Febiger, 1945, p. 281.

The conservative treatment of periodontal disease

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In the treatment of periodontal disease, there are many operators who believe that once the periodontal tissues are detached from the root surface, reattachment will not occur;[1,2] therefore, the overlying gingival tissues should be excised to the point of their remaining attachment. This is known as the surgical method of treatment. On the other hand, there are those who believe that the supporting tissues will return to normalcy and reattchment will occur by the use of conservative methods of treatment.[3-6] This latter group feels that surgical measures should be resorted to only after conservative measures fail.

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1. Black, A. D. Treatment of peridental inflammation coordinated with physiological reactions of the tissues. J.A.D.A. & D. Cosmos 24:1587 (Oct.) 1937.

2. Blanquie, R. H. Rationale of pocket elimination in the treatment of periodontal disease. J. Periodont. 21: 139 (July) 1950.

3. Bell, D. G. A case of reattachment. J. Periodont. 8:30 (Jan.) 1937.

4. Beube, F. E. Study of reattachment of the supporting structures of the teeth. J. Periodont. 18:55 (April) 1947.

5. Bjorndahl, O. Reattachment and bone regeneration: report of case. J.A.D.A. 36:356 (March) 1948.

6. Bunting, R. W. Oral hygiene and the treatment of perodontal diseases. Philadelphia, Lea & Febiger, 1936.

Each method has its advantages and disadvantages. The surgical method is more direct in its application and requires a shorter course of treatment. It involves only surgical technics which are familiar to the dentist. It is also much more spectacular. But its end result is not as favorable as the one achieved by the conservative method since the surgical method requires amputation of gingival tissues which usually are not restored. The conservative method, when successful, allows the periodontal lesion to heal without the loss of tissue. However, it requires a specific technic by which the involved areas may be cleansed by well directed instrumentation. This requires study and practice in order to be thoroughly executed.[6]

Many conscientious operators using conservative methods of treatment have seen clinical evidences of cure and reestablishment of lost gingival attachment. Their observations are substantiated by scientific investigations,[9,10] the latest and perhaps most significant of which are the findings of Ramfjord.[11] To those who have successfully treated periodontal disease and have seen their patients' mouths remain healthy over a long period of years, there is no question as to the value of conservative treatment.

The rationale of conservative treatment is based on the assumption that most periodontal disturbances arise from local irritation of the supporting tissues and on the observation that when these local irritants are completely removed, the tissues usually return to health. The major sources of irritation are accumulations of calculus, bacterial irritation, food impactions, faulty restorations and occlusal trauma.

Local irritation of the gingival margins leads to inflammatory processes, which involve the supporting tissues to a greater or lesser degree. When the teeth are kept continuously clean and function is maintained within physiologic limits, the gingival tissues are not subjected to injury. These tissues are usually firm and resistant. Strict oral cleanliness, then, is a most effective protective measure against periodontal disease.

Conservative treatment includes all the procedures which are contained in the broad definition of oral prophylaxis;[6,12] namely, the removal of all calcareous deposits and stains, the smoothing and polishing of tooth surfaces and fillings, the removal of overhanging fillings and other faulty restorations, and the relief of undue stresses on the teeth as well as the instruction of the patient in home care. Conservative treatment also includes special attention to those pockets which remain after the above procedures have been accomplished.

In all of the procedures involved in the conservative care of the mouth, the removal of all calculary deposits and debris from subgingival portions of the teeth is a most critical problem. It is easy to remove calculary deposits from the exposed surfaces of the tooth, but the care of the subgingival areas is a much more difficult matter. Unless these areas are properly cleansed, the gingival tissues cannot be healthy.

CARE OF SUBGINGIVAL AREAS

Care of the subgingival areas, even in normal and near normal mouths, is essential to the maintenance of mouth health. It requires an ability to explore and cleanse, by instrumentation, areas which are hidden from view. Since

7. Goldman, H. M. Subgingival curettage, a rationale. J. Periodont. 19:54 (April) 1948.

8. Leonard, H. J. Periodontal treatment for maximum reattachment. J.A.D.A. 22:602 (April) 1935.

9. Linghorne, W. J., and O'Connell, D. C. Studies in the regeneration and reattachment of supporting structures of the teeth. I. Soft tissue. J. D. Res. 29:419 (Aug.) 1950.

10. Box, H. K. Treatment of the periodontal pocket. Toronto, University of Toronto Press, 1928.

11. Ramfjord, S. Experimental periodontal reattachment in rhesus monkeys. J. Periodont. 22:67 (April) 1951.

12. Coolidge, E., and Hine, M. K. Periodontia. Philadelphia, Lea & Febiger, 1951.

thorough subgingival care will not only cure many periodontal lesions, but also prevent them, it should be practiced by every dentist. It is just as much a part of dentistry as are operative procedures.

In order to accomplish this, the dentist must develop a technic of instrumentation which will afford absolute control and yet preserve a keen tactile sense. The instrument should be held by the thumb and index and middle fingers, as the pen is held in the Spencerian type of writing.[13] This grasp is preferred because by this three point contact the instrument can be held and directed with a minimum of finger pressure. When only a two point contact (thumb and index finger) is used, greater pressure is required to hold the instrument. Tightness of grasp produces a numbing of the fingers and destroys the tactile sense which is so essential.

In propelling the instrument, the fingers should not be moved. They should grasp the instrument comfortably and continually maintain that position. Motion is accomplished by rocking the wrist or arm to actuate the cutting edge of the instrument, using the third finger as a fulcrum or rest. In this manner, precision and effectiveness of motion may be attained without loss of tactile sense. If the fingers are continually flexed, tactile perception is soon lost.

There is a wide range of instruments which may be used in conservative treatment. There are some sets of scalers made with a specific blade for each surface of every tooth. The operator who uses such a profusion of instruments spends far too much time at each appointment picking up instruments, using them for a stroke or two, and then laying them down. It is suggested that a few, fine, sharp scalers and files, with light balanced handles are much more practical. These instruments should be those designed to be used with a draw stroke in order to minimize tissue injury. Fine, blunt-pointed explorers also are needed to estimate the depth of pockets and to detect the roughness or smoothness of the root surfaces.

CHOICE OF TREATMENT

At the first examination of the patient, it is not always possible to determine whether conservative or surgical methods of treatment should be utilized. Some conditions which appear hopeless respond amazingly well to conservative treatment. In other instances, there may be a poor response, and more drastic treatment will be required. Since it is desirable to retain as much gingival tissue as possible, conservative methods should be instituted first. If the treatment is well done, there will be immediate improvement in the majority of cases, and if the mouth is kept clean, this will be followed by complete healing. Surgical treatment should not be resorted to until the mouth is put in a state of thorough cleanliness. When this has been done, many areas that looked hopeless will have healed. The less severe infections will have entirely disappeared.

TREATMENT

It is important to follow a definite order when treatment is begun. That is, the upper right or left posterior teeth should be treated first and then an orderly pattern is followed until all teeth have been covered. When the deposits are heavy, it is preferable to scale only a few teeth, or one quadrant of the mouth, thoroughly scaling each tooth, rather than to cover all the teeth superficially. At the next appointment, another quadrant can be treated with a rechecking of those previously done. Better results will be attained by treating the case two or three times a week, at first, rather than once a week or once in two weeks.

13. Bunting, R. W. Textbook of oral hygiene and preventive dentistry. Philadelphia, Lea & Febiger, 1950.

Some operators prefer to gain visual access to the subgingival deposits to facilitate their removal. For this purpose, they use mechanical packs in deep periodontal pockets. These may be made by rolling wisps of cotton in a thick paste, the chief ingredients of which are zinc oxide and eugenol. Pellets of various sizes may be pressed firmly into the pocket and left for 48 hours or four or five days. When the packs are removed, the soft tissues are retracted, there will be less bleeding, and space is afforded to gain better access to the denuded root surface. These packs are useful in the management of hyperplastic gingivitis. They are also used by many operators in the preparation of deep interproximal cavities. Care should be used, however, not to leave them in place too long or too much atrophy will result, causing permanent retraction of the tissues.

After each scaling, the teeth should be polished to reduce infective activity and the tendency to subsequent accumulations. The polishing may be done manually or with motorized equipment, or by a combination of both methods. At the conclusion of each treatment, some bland solution such as iodine lotion,[6] or iodosaline,[12] should be applied to the tissues by means of cotton pellets or a syringe. The majority of scaling and polishing should be completed in four or five appointments. At that time, some, or all areas, will show promise of healing, depending on the severity of the case, but there may be some which show unsatisfactory response. When all steps in the prophylactic procedure have been completed, most areas will have healed, but if there are some which still show an unsatisfactory response, a decision must be made as to whether the residual pockets shall be treated by surgical methods or whether further subgingival curettage should be carried out. This decision will be based on many considerations, such as the area of the mouth in which the pockets are present; whether esthetics is important; the depth and accessibility of the pockets; whether inflammation still persists, and whether the areas can be reached for home care by the patient.

If it is decided to continue conservative treatment, medicated packs should be used as an aid in subgingival curettage. The effect of these packs is to cauterize any epithelial lining which may be present and which would interfere with subsequent healing. These medicinal packs should not be used until the root surfaces have been thoroughly scaled so that the resultant clot may be left undisturbed for the healing process. Packs described by Barkann[14] are useful because they are easily managed. They are made of short lengths of seine twine which have been saturated in 75 per cent pure phenol and 25 per cent gum camphor. They should be pushed deeply and firmly into the bottom of the pocket and allowed to remain for five minutes. As soon as they are removed, the area should be rinsed with warm water. Curets and files are then used to smooth the cementum and remove the cauterized epithelium. The clot which results from this subgingival curettage should be undisturbed and isolated for about 10 minutes. The patient should be advised to rinse the mouth frequently with saline solution after a 24 hour period and that toothbrushing may be resumed in three or four days. Supervision must be continued at frequent intervals until the patient is able to maintain mouth cleanliness with only an occasional prophylaxis. Otherwise new accumulations of debris may militate against the healing process.

These conservative and prophylactic technics are exacting and can only be mastered by practice and close application to details. Indeed they should be learned in school. Adequate time should

14. Barkann, Lillian. A conservative surgical technic for the eradication of a pyorrhea pocket. J.A.D.A. 26:61 (Jan.) 1939.

be allotted in the curriculum for manikin practice in order that the students may learn the technics of scaling and polishing before they begin their clinical experience.

Students should be required to perform a thorough and complete prophylaxis for each of their clinical patients before they begin operative procedures. They should do this frequently enough so as to see the benefits of such treatment. They should demonstrate an ability to treat by conservative measures cases of gingivitis, Vincent's infection and periodontitis before they are allowed to use surgical procedures.

HOME CARE OF THE MOUTH

Regular home care of the mouth is of great importance in the maintenance of mouth health and in the prevention of gingival disturbances.[15,16] It is also an essential part of the treatment of periodontal diseases. The majority of dentists, however, do not take time to teach their patients how to take care of their mouths. Consequently, most people are neglectful of their mouth care. Perhaps this failure on the part of the dentist springs from a lack of uniformity of thought as to what should be taught and practiced in home dental care. In reviewing the literature one is struck by the variety of methods suggested and the uncompromising attitude of many of their proponents.

One writer decries the fact that children are not given training in toothbrushing as a part of the school curriculum. Then he goes on to describe what he thinks should be taught and states that several weekly appointments are necessary to master his recommended technic.

Some propose methods which are not suitable for children. Others recommend methods adaptable where there has been extreme recession or for postoperative care but methods which are not proper for normal mouths. Some brush up, while some brush down, and some brush in circles. Some insist on soft brushes, some medium and some extremely hard. Is it any wonder that the average dentist is confused, to say nothing of the public?

Since it is known that toothbrushing is valuable and should be practiced by all people, then there is need for some method of mouth cleansing that is not only easy to teach but one that can be readily used by the average person. It should be one that will produce the minimum amount of injury and one that requires the minimum time and equipment.

No matter how idealistic a dentist may be or how convinced that some complicated and detailed method of brushing will work wonders in the maintenance of mouth health, he must also be realistic enough to know that only a small percentage of the population who have the individual attention of a dentist will be taught these methods. The average citizen brushes his teeth rapidly just before his morning coffee. At noon, he has no brush to use after his lunch, and at night he may give his teeth a few perfunctory strokes. It will be difficult to correct his toothbrushing habits unless he is given some simple method; one so simple and direct that he will be inclined to adopt it as a daily habit.

A method of toothbrushing which is widely used today may be called a sweeping stroke, or rolling stroke, carrying the brush down on the upper teeth and up on the lower teeth. This is a method which can be easily taught and practiced. It can be used by adults and by children, in healthy or in diseased mouths. It can be used on the lingual as well as the buccal surfaces. It can be executed with toothbrushes which have soft, hard, nylon, or natural bristles. There are a few simple but important directions,

15. Hine, M. K. Use of the toothbrush in the treatment of periodontitis. J.A.D.A. 41:158 (Aug.) 1950.

however, which the patient must follow in order to make this method effective.

First, the patient must watch each stroke in the mirror. Most people brush their teeth in front of a mirror, but unless they are told to look at their teeth and gums as they brush them, they incline their heads forward and look in the wash bowl. They depend on feeling rather than sight. The operation can only be performed successfully if the patient's eyes are open and he watches what he is doing. Those who wear glasses for reading should put them on when they brush their teeth.

Second, the patient should place the brush against the upper gingival tissue with the bristles pointing up, and should exert pressure on the tissues with the side of the bristles to blanch them momentarily. This will tend to stimulate the gingival circulation and increase tissue resistance.

Third, he should move the brush by a wrist motion carrying the bristles downward and slightly laterally in order to engage the interproximal surfaces as well as the cervical areas. This wrist action is extremely important in making the brushing effective. Without it, the bristles tend to jump from the gingival tissue to the convexity of the crowns, thereby skipping the cervical region. On the lower teeth the bristles should be carried upward in a reverse manner to that used on the upper teeth. The strokes should be performed slowly and repeated four or five times in each position until all areas of the mouth have been covered. In this, there should be a definite pattern, beginning on the right or left posterior area and continuing in an orderly manner until every tooth has been reached.

Photographs and models often will serve to impress patients with the differences between the healthy, pink, stippled gum tissues and those which are in various stages of inflammation. Then, if they will watch the change in color of their own tissues and note the improvement of their mouths under proper home care, they will be more inclined to adopt such measures as a permanent habit.

TOOTHBRUSHES

The selection of the brush should be made in accordance with the individual needs of each patient. Not all should use stiff black-bristled brushes, nor should all use soft ones. In general, it may be said that a stiff, two or three row brush, with a rather rigid straight handle is best for most people. Bristle stiffness may be varied by the use of more or less water before using. The brush should be approximately six and a half inches long with a one inch head. The bristles should be three-eighths to a half inch in length, arranged in pointed tufts. Handles of different color serve to distinguish which is to be used morning, noon and night. There is no advantage in twisted or angulated handles over a straight handle. In fact, some makes of brushes would be definitely improved if the head were set on a straight instead of an angulated handle.

CARE OF THE TOOTHBRUSH

As a result of their study of toothbrushes in use in American homes, Gruebbel and Wisan[16] were impressed with the unsanitary condition of most people's brushes and concluded that more emphasis should be placed on their care. People should be taught to use them for brushing the teeth and not for cleaning silver or shoes.

Too many people possess only one toothbrush and that is so battered and soggy that it is no longer of any use for cleaning the teeth. They should have one toothbrush for each time in the day

16. Gruebbel, A. O. and Wisan, J. M. A study of toothbrushes in American homes, J.A.D.A. 37:346 (Sept.) 1948.

that they clean their teeth. If they brush them after each meal, they should have three brushes. At the least, mouth care should be given twice daily, which would require two brushes to be used alternately. This will allow a 24-hour drying period for each brush. Thorough drying will make it a cleaner and more effective instrument for stimulating and brushing.

After using the brush, it should be held under running water until all the food and dentifrice are washed out. It should then be stood in an open container, such as an empty water glass, or it should be hung where it will dry in the air. Brushes cared for in this manner and used only once daily, should last four or five months.

Some people believe that their toothbrushes are not clean unless they are kept immersed in some antiseptic solution. Such immersion makes the brush useless for the purpose for which it is intended.

DENTIFRICES

Dentifrices have no therapeutic value. They are, however, useful aids in polishing tooth surfaces. It has been suggested that a good dentifrice should consist of equal parts of water and elbow grease.[17] This may satisfy those who like water and elbow grease, but the majority of persons prefer a more pleasing flavor. For most people, brushing the teeth is an unpleasant, time-consuming task. If a flavored dentifrice makes toothbrushing more acceptable, it should be used. Soap and mild abrasives are helpful in removing dirt from the hands. They are also useful in removing debris from the teeth.

The problem of selecting a dentifrice which one can recommend has become increasingly difficult since the Council on Dental Therapeutics no longer lists those which are approved. Unless the patient is using a dentifrice which is known to be highly abrasive, it is probably wise to allow him to continue to use the one he likes best. Robinson and Kitchin[18] reported that in a series of dentifrices tested, the majority of pastes were less abrasive than powders. Those which contain sugar are definitely contraindicated.

DENTAL TAPE

Dental tape should be used to remove debris which may become wedged between the teeth. It is not an interdental stimulator and should not be used as such. In applying the tape, it should be held between the thumb and forefinger of each hand with the shortest segment possible. It should be carried between the teeth with a slow, sawing motion. When a long segment is applied, it is apt to snap between the teeth producing injury to the interdental tissues. The flat ribbon tape is preferable to the threadlike type. The patient should always watch in the mirror as he uses it. This will enable him to apply it much more efficiently with less danger of cutting the tissues. As a rule, children up to the age of 14 do not need to use tape. In fact, they may do more harm than good by using it hurriedly and carelessly.

INTERDENTAL STIMULATORS

Interdental stimulation, when indicated, may be accomplished by the use of properly shaped wood or rubber points. These are specially designed to fit the interproximal spaces and to be pushed through them as far as they will go comfortably. Most people learn the use of these applicators more readily than interdental brushing. Their use, too, is facilitated by looking in the mirror.

The rubber stimulator is mounted at

17. Fenton, H. R. Toothpastes, powders, brushes, and mouthwashes. North-West Den. 23:41 (Jan.) 1944.

18. Kitchin, P. C., and Robinson, H. B. G. How abrasive need a dentifrice be? J. D. Res. 27:501 (Aug.) 1948.

an angle which permits the tip to be introduced from the lingual as well as the labial aspects. These stimulators not only massage the interproximal tissues, but also remove plaques and accumulations of food between the teeth.

The so-called "Stimudents" may be used in the anterior part of the mouth, but are not so well adapted to the posterior region. If toothpicks are used for this purpose, they will do less damage if they are flat and not round.

MOUTHWASHES

Mouthwashes, like dentifrices, have little, if any, therapeutic value. The mouth should be rinsed after brushing the teeth to flush out any remaining debris, and the least expensive and most available wash is water. Some recommend salt and soda while many patients like to use the more highly flavored preparations. Unless they are too highly loaded with astringents, they do no harm and may leave a flavor in the mouth that is refreshing and reconciles some people to brushing their teeth.

Hydrogen peroxide, 3 per cent, diluted with equal parts of water may be used on the brush occasionally to aid in cleansing the teeth. It also may be used on the tongue to cleanse it from accumulated debris. It should be applied to the tongue with a soft brush, not a stiff one, in a light stroke sweeping from the posterior to the tip. It has been frequently found that many cases of persistent halitosis may be relieved by an occasional brushing of the tongue with peroxide.

SUMMARY

In this discussion of the conservative treatment of periodontal disease and the home care of the mouth, it has been possible to state only the general principles involved. Many details and important correlated factors have necessarily been omitted.

Two most important features have been accentuated; one, that when all local irritants have been removed from diseased periodontal tissues the disease will be arrested, the lesions will heal and normal tissue relationships will be restored in a large percentage of cases.

Second, when mouths are kept clean enough, especially the subgingival areas, the periodontal tissues usually will be maintained in health indefinitely. Adequate prophylactic measures and well directed home care, therefore, constitute a most effective protection against periodontal disease.

Dentists have been too much concerned with the rehabilitation of mouths wrecked by periodontal disease and have not given enough attention to the prevention of such destruction.

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Surgical treatment of periodontal disease

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The elimination of the periodontal pocket, utilizing surgical methods, is a much more spectacular and rapid technic than the conservative methods of curettement. The purpose of this paper is to evaluate the various technics and the rationale of surgical treatment of periodontal disease. However, surgical treatment alone will not eliminate the inflammatory process from the gingiva permanently. It is only one of the aids that are available in the attempt to eliminate this pathologic process.

Judging from a review of available literature on this subject of surgical treatment, there seems to be some dispute about the first dentist to introduce surgical procedures in periodontal therapy. Leonard[1] states that surgical resection was advocated in France by Fauchard in 1728. Merritt[2] credits Kunstmann, who published a small book in 1772, as one of the first to attempt gingivectomy. Zentler,[3] on the other hand, stated that Robicsek, a Hungarian, in 1894 advocated surgical treatment by a form of flap operation in which access to the area for curettement was attained without actual removal of the pocket. This paper will trace the development of surgical procedures from 1912 only.

TWO DIFFERENT APPROACHES

Basically, there are two distinctly different approaches to the eradication of periodontal pathology by surgical methods: (1) the gingivectomy or complete removal of the gingival tissues forming the pocket;[4] (2) the so-called flap operation

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1. Leonard, H. J. Appraisal of developments in periodontology from 1901-1950. J. Periodont. 22:107 (April) 1951.

2. Merritt, A. H. Surgery's place in the treatment of periodontoclasia. J.A.D.A. 29:771 (May) 1942.

3. Zentler, Arthur. Suppurative gingivitis with alveolar involvement. A new surgical procedure. J.A.M.A. 71:1530 (Nov. 9) 1918.

4. Goldman, H. M. Periodontia: a study of the histology, physiology and pathology of the periodontium and the treatment of its diseases, ed. 2. St. Louis, Mosby, 1949, p. 411.

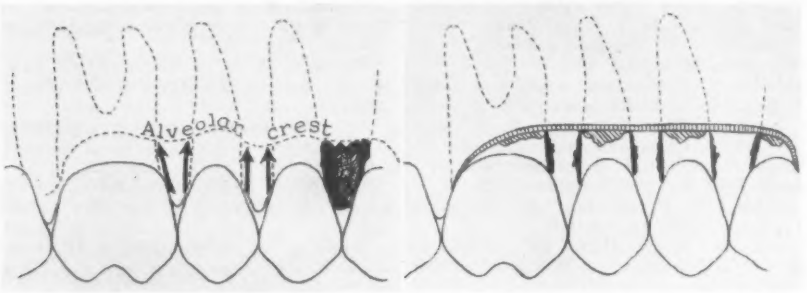

Fig. 1 • Ward operation. Left: Schematic representation of an area that has been obliterated with the interproximal file. The file was carried through the soft tissue until bone was contacted. Right: A horizontal incision was made buccally and lingually to facilitate removal of the remaining tissue. Calculus and bone were apparent after removal of the soft tissue

which consists of the elevation of a flap of soft tissue to gain access to the diseased area for curettement.[5]

Madison[6] reported that the first detailed surgical technic was introduced in 1912 by an Englishman named Pickerill. He advocated the removal of all calculus and the final excision of all infected soft tissue. Applications of tincture of iodine were made frequently to the wound surface. But Black,[7] in 1915, was the first recorded American investigator to describe gingivectomy. He felt that the gingiva should be excised to the bottom of the pocket, or a little beyond, which involved the removal of a portion of the alveolar process. He stated:

> In cases in which the pockets on the labial, buccal, or lingual surfaces are deep, the depth may be reduced by cutting away the overlying tissue, or in other words by cutting away the gum tissue that is undermined by the disease of the periodontal membrane.

Yet Black went on to advocate the removal of a portion of the alveolar process, but this unnecessarily destroys tissue which can be restored to health. He also suggested the use of cautery to make the excisions and cited control of hemorrhage as the principal reason for its use.

Slight modifications of the technic of gingivectomy were made in England by Nodine,[8] in 1920. That same year, in Philadelphia, Ziesel[9] introduced "gingivoectomy," and described a clear outline for the removal of the soft tissues forming the pocket which was to be followed by curettement of necrotic bone.

Ward,[10] in 1928, advocated the removal of periodontal pathology by treatment which he termed the "obliteration" method. This treatment consisted of the removal of all of the interproximal tissues down to and including portions of the alveolar process which, Ward stated, was permeated with bacteria. (See Figure 1, left.) His removal of the soft tissues was unique in that it was accomplished by the use of interproximal files. These files were thrust through each affected interproximal space and with pushing strokes all tissue was eliminated from

5. Goldman, H. M. Periodontia: a study of the histology, physiology and pathology of the periodontium and the treatment of its diseases, ed. 2. St. Louis, Mosby, 1949, p. 434.

6. Madison, W. J. A review of some surgical methods of treating pyorrhea. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 25:898 (Sept.) 1939.

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8. Nodine, A. M. Surgical treatment of pyorrhea. D. Cosmos 63:345 (April) 1921.

9. Ziesel, William. Pyorrhea extermination: gingivoectomy. D. Cosmos 63:352 (April) 1921.

10. Ward, A. W. Surgical eradication of pyorrhea. J.A.D.A. 15:2146 (Nov.) 1928.

the region. These cuts were then joined by a horizontal incision and the buccal and lingual flaps of remaining tissue were removed (Fig. 1, right). Ward's radical treatment may be questioned, but he made a valuable contribution to surgical resection by his introduction of a cement that could be applied over the operative site during the healing period and thus prevent unnecessary discomfort.

The following year Kaiser[11] made a slight variation of the method for removal of the pocket. Vertical incisions were made in each affected interproximal space to the depth of the pocket. These parallel incisions then were connected in a horizontal plane and the soft tissues removed. This removal was followed by a smoothing of the alveolar crest, the removal of the necrotic bone and the scaling of the root surfaces. The entire operative site was covered by a surgical cement until healing was complete.

In 1932, Crane and Kaplan[12] introduced a technic which claimed to preserve the maximum amount of normal tissue. A pocket-marking forceps was introduced to the depth of the pocket

11. Kaiser, F. S. The surgical treatment of pyorrhea alveolaris. J.A.D.A. 16:1299 (July) 1929.

12. Crane, A. B., and Kaplan, Harry. The technique and results of surgical pyorrhea treatment. D. Digest 38:3 (Jan.) 1932.

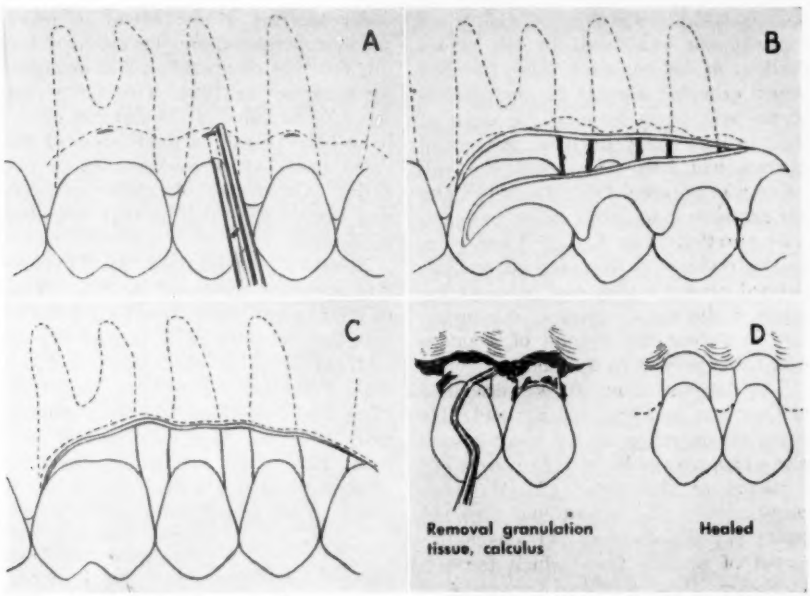


Fig. 2 • Crane-Kaplan pocket marker and operation. A: Pocket marker introduced to the depth of the pocket and the handles compressed to leave a mark on the attached gingiva. B: Removal of the strip of soft tissue. Calculus can be seen on the surface of the roots. C: Operation completed and calculus removed. The bone has been reshaped and tissue tags detached. The area is ready for a surgical cement dressing. D: Modified Crane-Kaplan operation. Careful removal of excessive granulation tissue and calculus is seen at the left. The area after healing is shown at the right

(Fig. 2, A) and compression of the handles left a bleeding point on the surface of the free gingiva. An incision was made connecting the bleeding points and the strip of diseased tissue was removed with a blunt dissector (Fig. 2, B). Interproximal saws were used to break down the diseased bone. Calculus then was removed from the necks of the teeth, the bone shaped to the proper form and smoothed and unsupported tissue tags were removed (Fig. 2, C). The area then was covered with surgical cement and left until healing had been accomplished.

However, in May, 1951, Kaplan and Milobsky[13] modified the original Crane-Kaplan technic. The basic change was made to conserve bone, connective tissue and periodontal fibers. The depth of the pockets on the buccal or labial and lingual surfaces was established by the pocket marker, as before, since these bleeding points provided a guide for the excision of the soft tissue forming the walls of the pocket. When the two sections of gingiva had been eliminated, the calculus was removed from the roots and the excessive granulation tissue was carefully curetted (Fig. 2, D). These men, in the revision of the original technic, stressed careful scaling and thorough hygiene of the mouth prior to the operation to reduce the amount of granulation tissue present in the pocket.

The flap operation first was described in Germany by Cieszynski in 1914.[6] He based his operation on the seven principles which may be listed: (1) convenient entrance to the area; (2) thorough curettement; (3) smoothing crenated bone; (4) disinfection; (5) radical removal of pockets from which there is exudation; (6) removal of the epithelial lining of the pocket; (7) use of steel instruments, not galvanocautery. However Zentler,[3] in 1918, was the first to publish a description of the flap operation in America. An incision at the free gingival margin was carried to the apexes of the teeth. A flap was elevated to include the periosteum and, while the flap was retracted, "all of the inflamed, infected and granulomatous tissue" was removed (Fig. 3, left). With chisels and a mallet, the alveolar plate was removed from around the denuded roots to insure the removal of the "infected bone." The rough edges of the remaining bone were smoothed and, following the removal of the granulation tags on the inner aspect of the elevated flap, sutures were placed to close the wound (Fig. 3, right). The entire area then was covered with iodoform gauze. The writer provides no description of a method of treatment for pockets that were lingually located, and limited the operation to three or four teeth to avoid so-called "septic absorption."

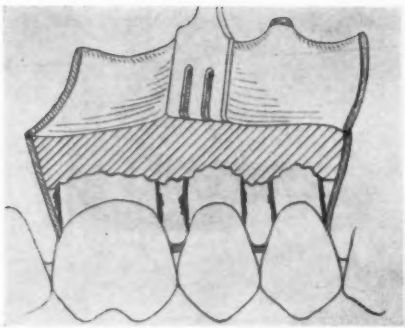
A reduction in the extent to which the mucoperiosteal flap was elevated during this type of operation was described by Shearer[14] in 1924. This more conservative handling of the flap and underlying bone permitted more areas of diseased tissue to be operated on at one sitting. Otherwise, Shearer's operation was exactly the same as that described by Zentler.

Zemsky's modification of Shearer's flap operation appeared in 1926.[15] One of the changes which Zemsky introduced was the elevation of a lingual flap in addition to either the buccal or labial flaps. Following the curettage and bone filing and smoothing, the free gingival margins of the flaps were excised to a point that when sutured together the newly-contoured alveolar crest was exposed. This minor variation was per-

13. Kaplan, Harry, and Milobsky, Louis. A surgical procedure for periodontal pocket elimination: revised Crane-Kaplan technique. Oral Surg., Oral Med. & Oral Path. 4:546 (May) 1951.

14. Shearer, W. C. So-called surgical treatment of pyorrhea alveolaris. Int. J. Ortho. & Oral Surg. 10:731 (Nov.) 1924.

15. Zemsky, J. L. Surgical treatment of periodontal diseases with the author's open-view operation for advanced cases of dental periclasia. D. Cosmos 68:465 (May) 1926.



Right: The operation completed and sutures in place

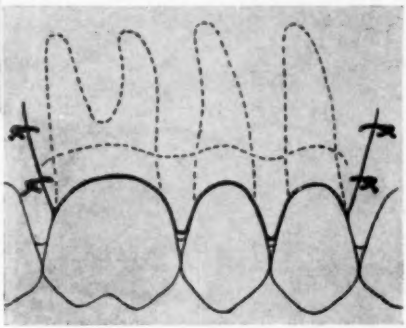


Fig. 3 • Zentler's flap operation. Left: Mucoperiosteal flap retracted. Note height of incision was not to the apexes of the teeth as was advocated by Zentler

formed on the premise that all of the infected tissues thereby were removed.

The most widely cited type of flap operation was introduced in 1931 by Kirkland[16] and was called the modified flap operation. This technic was advocated for purulent lesions about single-rooted teeth, and also anterior teeth where gingivectomy would be contraindicated. An incision was made in the interdental papillae to the depth of the pocket and then carried mesially and distally separating the crest into two parts so that the flaps could be retracted both labially and lingually (Fig. 4, right). While the flaps were held retracted the root was scaled to remove calculus and the area was curetted to remove granulation tissue and the crenated bone was smoothed. Adhering granulomatous tags on the inner aspect of the flaps also were removed. The flaps were coaptated and sutured and a wax dressing was applied to the area until healing was accomplished in from three to five days (Fig. 4, left).

In 1936, Kirkland described his semi-flap technic in which the modified flap just discussed was utilized on the buccal or labial aspects of the pocket but with a complete gingivectomy on the lingual surface. He developed this modification because, as he stated, the patient was prone to neglect the lingual surfaces of the gingivae and hence the areas of infection were more deeply seated.[17]

A conservative surgical technic was outlined by Barkann[18] in 1939. She devised this operation for the removal of the contents of the pocket including the epithelial lining. She stated that the operation was performed "so that contact between the root and the gingivae, in their normal relationship may be re-

16. Kirkland, Olin. The suppurative periodontal pus pocket; its treatment by the modified flap operation. J.A.D.A. 18:1462 (Aug.) 1931.

17. Kirkland, Olin. Surgical flap and semiflap technique in periodontal surgery. D. Digest 42:125 (April) 1936.

18. Barkann, Lillian. A conservative surgical technique for eradication of a pyorrhea pocket. J.A.D.A. 26:61 (Jan.) 1939.

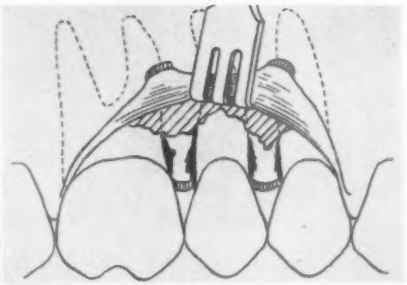
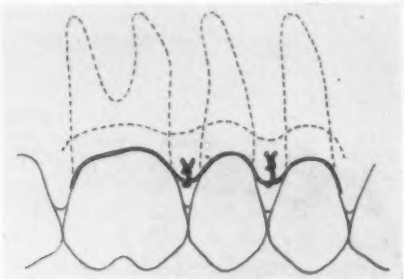


Fig. 4 • Kirkland's modified flap operation. Right: After incising the interdental papillae the flaps are retracted buccally and lingually to gain access for the removal of calculus and granulation tissue and for smoothing the bone



Left: Interdental sutures have been placed, and are allowed to remain until healing has been accomplished

established." This procedure did not involve the removal of any of the buccal and lingual gingivae because the writer did not feel that they were infected and also because these tissues served as a natural support for the blood clot (Fig. 5).

The chief accomplishment of gingivectomy in periodontal practice is the complete elimination of the pocket and re-establishment of a shallow crevice. This operation removes all of the soft tissue forming the walls of the pocket down to the epithelial attachment. Immediately beyond the pocket is connective tissue, in a state of inflammation, along with transseptal fibers of the periodontal membrane, the alveolar bone and the alveolar process. Fish,[19] Kerr,[20] Orban[21,22] and others,[13,23,24,25] on the basis of histopathologic studies, have established that there is no infectious process present in the supporting bone. This fact is directly opposed to the opinion of the men who were responsible for the development of the various surgical technics used in the treatment of periodontal disease. Orban[22] states that the inflammatory reaction may penetrate the marrow spaces of the supporting bone, but this bone is neither infected nor necrotic. In certain instances, however, when the base of the pocket is apical to the alveolar crest, bone must be removed to eliminate the pocket. All of the previous proponents of the radical flap operation appear to have violated this basic fact when they advocated the smoothing and curetting of the infected bone. All flap operations should not be condemned, because they have a definite place in periodontal therapy, but from sound histopathologic evidence, curettement should not extend beyond the apical proliferation of the epithelial attachment. Bone must be removed or

19. Fish, E. W. Bone infection. J.A.D.A. 26:691 (May) 1939.

20. Kerr, D. A. Personal communication, June, 1951.

21. Orban, Balint. Gingivectomy or flap operation? J.A.D.A. 26:1276 (Aug.) 1939.

22. Orban, Balint. To what extent should the tissues be excised in gingivectomy? J. Periodont. 12:83 (July) 1941.

23. Goldman, H. M. Gingivectomy: indications, contraindications and method. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 32:323 (May) 1946.

24. Saghirian, L. M. Electrosurgical gingivoplasty. Oral Surg., Oral Med. & Oral Path. 2:1549 (Dec.) 1949.

25. Kronfeld, Rudolf. The condition of the alveolar bone underlying periodontal pockets. J. Periodont. 6:22 (Jan.) 1935.

recontoured on occasion to establish proper gingival architecture.

Figure 6 illustrates the extent to which the excision of the soft tissues should be carried, on the basis of pathology. From the diagram, the line of excision is at once apparent and the location of the line is at the base of the sulcus. The underlying connective tissue, which has a certain amount of inflammatory reaction present, need not be curetted because to do so would destroy regenerative elements and would remove unnecessarily those tissues which would become normal[21, 26] when the inflammation subsides. If large masses of granulation tissue are present, they may be removed gently, without disturbing the underlying connective tissue. The excess granulation tissue is removed to prevent the re-formation of a gingival pocket and to increase the rate of healing.[26]

HEALING

Two excellent pieces of research have been completed, the first by the pathologists Orban and Archer,[26] to explore the process of wound healing following gingivectomy. The first study was reported in 1945. Orban and Archer completed their experiment by studying microscopically a granulating wound surface at serial intervals, and found that complete epithelialization did not occur until the fourteenth postoperative day. Their study was completed without the application of surgical cement dressing to the area. The conclusions drawn from their experiment were that the chronic inflammation present in the deeper layers of the gingivae was not altered by the operation and that this tissue persisted following the gingivectomy. This granulation tissue then should be treated carefully so that it can be transformed into active mesenchymal tissue capable of regenerative activity. However, if this granulating mass is not kept controlled, it becomes excessive postoperatively and a gingival pocket will recur.

Two years later, Bernier and Kaplan[27] conducted a series of examinations similar to those of Orban and Archer, the only difference being that a surgical dressing was used to cover the wound. Their conclusions were that the presence of the pack increased the rate of healing. They demonstrated complete epithelialization in six days as compared to the earlier study in which the wound surface did not attain a complete epithelial covering until the fourteenth postoperative day. Bernier and Kaplan felt that the constituents of the pack were secondary to the presence of the pack on the

26. Orban, Balint, and Archer, E. A. Dynamics of wound healing following elimination of gingival pockets. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 31:40 (Jan.) 1945.

27. Bernier, J. L., and Kaplan, Harry. Repair of gingival tissue after surgical intervention. J.A.D.A. 35:697 (Nov. 15) 1947.

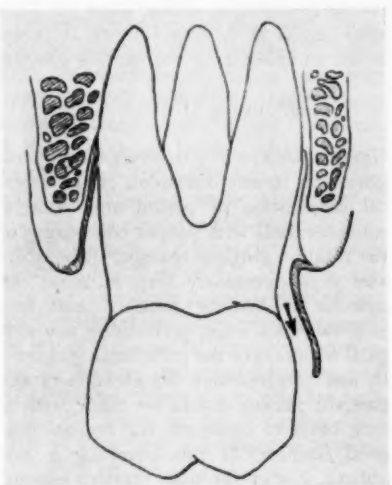


Fig. 5 • Barkann operation. The area on the left depicts the epithelium extending to the base of the intrabony pocket. The area on the right shows the crevicular epithelium being removed from the base of the pocket to the free gingival margin

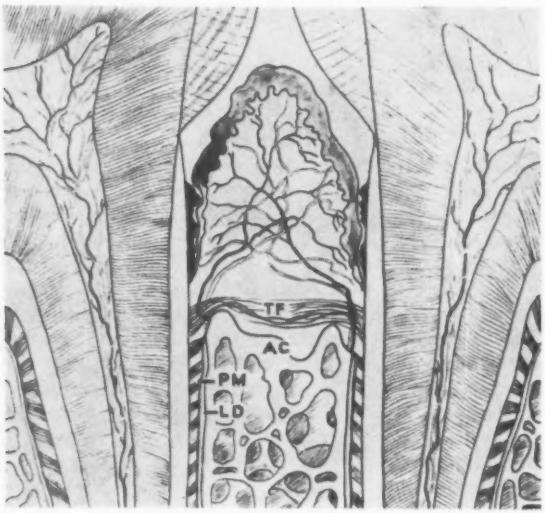


Fig. 6 • Schematic reproduction of periodontal pockets between two lower molars. Key to the letters is: TF, transseptal fibers of the periodontal membrane; PM, periodontal membrane; LD, lamina dura or alveolar bone; AC, alveolar crest. The area coronal to the transseptal fibers is heavily infiltrated with inflammatory cells. The line of incision for removal of the pocket would be from the base of the sulcus on the right to the base of the sulcus on the left

wound. These investigators also stressed the importance of the cautious removal of the granulation tissue, since its presence might be a most important factor in the stimulation of the healing process.

GINGIVOPLASTY

Gingivoplasty, which deserves special consideration in any discussion of the surgical elimination of periodontal pockets, concerns itself with proper contouring of the residual gingival margin.[28] The operator must constantly keep in mind the contour of healthy attached and free gingival areas when performing any surgical resection of the pathologic gingivae. In any gingivectomy the excision of the gingival pocket should be made with a long bevel to approach the normal gingival contour. If this bevelling is not utilized, a shelflike edge remains around the necks of the teeth on which Goldman[28] has observed impingement during mastication and the accumulation of food debris. Following excision of the periodontal pocket, the remaining interdental papillae must extend further in a coronal direction than either the buccal or lingual plates of bone. On occasion, in order to accomplish this contouring, it will be necessary to reduce the plates of bone to a point where a natural appearing interdental papillae can be attained. The bone is not involved by any specific pathology, but, in order to gain proper contour of the gingival margin, this bone must be carefully removed. If a flat or depressed interdental papilla is allowed to remain, food, debris, materia alba and bacteria will reinitiate the inflammatory process with repetition of the formation of a periodontal pocket.

TECHNICS

In the technics discussed in this paper, all operators have utilized a steel knife to resect tissue. However, Webb,[29] in

28. Goldman, H. M. Development of physiologic gingival contours by gingivoplasty. Oral Surg., Oral Med. & Oral Path. 3:879 (July) 1950.

29. Webb, G. F. Electro-surgical treatment of pyorrhea: preliminary report of a new method. D. Survey 11:15 (Aug.) 1935.

1935, introduced a method of eliminating the periodontal pocket by electrosurgical means. He designed a bipolar electrode that coagulated the tissue proteins between the two poles. Later, according to Saghirian,[24] clinical experience demonstrated that the heat developed between these two poles penetrated deeply and destroyed normal supporting structures. Saghirian[30, 31, 32] first started application of the high frequency currents for gingival resection in 1933. His first publication appeared in 1940.[30] He stated that the electrode remained cold during the operation, although a current was generated within the tissues which produced a destructive heat that caused coagulation of the proteins. When the instrument is properly controlled, this may be concentrated to a small area with a resultant incision and a fine coagulation along the line of cleavage. The extent of coagulation is dependent upon the resistance of the tissue (fat, cartilage and bone being highly resistant and vascular tissue being the least resistant), the density of the current and duration of application of the current. In 1949, Saghirian,[24] in describing the technic, stated that cutting and coagulating were dependent upon frequency, wave pattern, amount of current and size and shape of the electrode. The principal advantages of the high frequency electrode over steel instruments are a nearly bloodless field and much more finely controlled excisions, which is highly important in contouring and shaping the remaining gingival margin, so that a normal festooned gingiva is achieved.

Prior to any gingivectomy or surgical procedure the acute inflammatory process should be eliminated. The reduction in swelling decreases the depth of the pocket to some extent,[28] there is less granulation tissue present in the pocket and there is less hemorrhage during the operation.[13] This treatment is accomplished by scaling and polishing the teeth as many times as is considered necessary.

SUMMARY

It has been determined from histopathologic evidence that the removal of periodontal pockets does not entail the destruction of any connective tissue apical to the base of the pocket. For years the mode of surgical treatment was to remove the soft tissue forming the pocket and then curet, plane, smooth and destroy portions of supporting bone. It now is known that to excise the pocket to the bony crests removes certain basic elements in the connective tissue that are highly important to the reparative process.[26]

It also has been found that most of the advocates of the flap operation have destroyed periodontal fibers, connective tissue, and alveolar process. It may be repeated, however, that all flap operations are not predicated on the curettement or smoothing of the bone and are not to be ruled out as an adjunct in periodontal therapy.

It has become evident that proper gingival architecture must be kept in mind when re-establishment of a new free gum margin is undertaken, for, without proper festooning, reinfection is invited. It has also been learned that electrocoagulation is a valuable means by which gingival contouring can be accomplished and that this means, when properly controlled, permits the operator to obtain an almost bloodless field.

It has also been learned that proper postoperative care must be maintained, the use of the surgical dressing is important to increase the rate of healing and the excessive granulation tissue which frequently forms on the wound surface must be kept to a minimum to prevent recurrence of the pocket.

30. Saghirian, L. M. Electrosurgery in the mouth. D. Digest 46:20 (Jan.) 1940.

31. Saghirian, L. M. Electrosurgery and collateral treatment. D. Digest 46:312 (Sept.) 1940.

32. Saghirian, L. M. Periodontosis: treatment by surgical and electro-surgical gingivoplasty. J.A.D.A. 38: 67 (Jan.) 1949.

Relation of operative and prosthetic dentistry to periodontal disease

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It is a well recognized fact that operative dentistry affects the health of the periodontium. This effect is either favorable or unfavorable and is exerted on healthy or diseased periodontal tissues.

There are constant references in the literature to "faulty dentistry," as a major etiologic factor in periodontal disease, particularly in gingivitis and periodontitis. In fact, "faulty dentistry" is discussed[1-8] so much that one begins to wonder whether there is any operative dentistry correctly done.

This prevalence of faulty dentistry offers abundant proof that its relationship to periodontal disease still is unrecognized and its importance unrealized. Routine clinical observations and experience tend to substantiate these facts.

General dental practice consists of two equally important parts—periodontics and associated restorative service. A good philosophy of dental service would be directed to the treatment of these diseases in such a way that the natural teeth and their supporting structures would be retained in a healthy state for the lifetime of the individual and full dentures would thus be avoided.

The periodontal phase of preventive, curative and maintenance dental practice, has become a firmly established part of progressive dentistry. Its objective is to establish and maintain a healthy periodontium. Only on such a foundation can long-range, successful restorative procedures be based.[9] The work of Grubb,[10] Granger,[11] Branstad[12] and others supports this fact.

The restorative phase of dental practice consists of the individual restoration, the permanent bridge, the removable bridge and splinting procedures. The individual restoration is the basic unit in all these restorative services. Two or more units soldered to pontics form the permanent bridge. Units connected to saddle areas by attachment devices form the removable bridge. Units soldered together in multiple form splinting devices.

1. Black, G. V. Operative dentistry, vol. 4. Chicago, Medico-Dental Pub. Co., 1936, p. 112-127.

2. McCall, J. O., and Hugel, I. M. Movable-removable bridgework. Principles and practices as developed by H. E. S. Chayes. D. Items Interest 71:512 (May); 71:614 (June); 71:722 (July); 71:1046 (Oct.) 1949.

3. Bartlett, A. A. Some principles of modern fixed bridge construction. J.A.D.A. 29:2166 (Dec. 1) 1942.

4. Weaver, S. M. Maintenance of natural conditions by intermittent pressure supplied by movable-removable bridgework. D. Survey 22:48 (Jan.) 1946.

5. Robb, H. M. Practical periodontia, Toronto, University of Toronto Press, 1950.

6. Smyd, E. S. Metal inlays. In American textbook of operative dentistry, ed. 8. Gabel, A. B., editor. Philadelphia, Lea & Febiger, 1947.

7. Smyd, E. S. Advanced thought in indirect inlays and fixed bridge fabrication. J.A.D.A. 31:759 (June 1); 31:913 (July 1) 1944.

8. Hirschfeld, I. Individual missing tooth: a factor in dental and periodontal disease. J.A.D.A. 24:67 (Jan.) 1937.

9. McLean, D. W. Diagnosis and correction of occlusal deformities prior to restorative procedures. J.A.D.A. 26:928 (June) 1939.

10. Grubb, H. D. Occlusal reconstruction. J.A.D.A. & D. Cosmos 25:372 (March) 1938.

11. Granger, E. R. Bio-mechanics of periodontal disease. J. Periodont. 21:98 (April) 1950.

12. Branstad, W. Problem of function in periodontal treatment. J. Periodont. 21:105 (April) 1950.

This all-important individual restoration must be built to two sets of specifications; namely, periodontal and mechanical. The mechanical specifications are determined by the inner surface of the restoration. Its contacting surface is the cavity preparation of the tooth. Laws underlying basic requirements of cavity preparation were established long ago by Black.[13] Smyd,[7] in 1944, added the engineering principles involved in inlay and inlay bridge abutment retention. Details of this phase of construction seem to be fairly well understood and are applied with varying degrees of success.

The periodontal specifications of a restoration are determined by its outer surface. These specifications have to do with the shape and form of this surface as determined by contact, contour, occlusion, margins and polish. The management of these five factors determines the amount of protection afforded the supporting structures by the restoration.

Details of this phase of construction, for some reason, seem to be less well understood. Failure to fulfill these five periodontal specifications is the cause of vast amounts of periodontal disease.

Execution of these periodontal specifications for a restoration in a healthy mouth equals prevention and maintenance of health. In the presence of periodontal diseases, it equals cure and maintenance of a healthy condition. Failure to execute these periodontal specifications initiates disease under healthy conditions. Failure in the presence of disease means an accelerated rate of destruction of the supporting structures. This relation of cause and effect applies to all restorative procedures, as the individual restoration is the basic unit in all.

THE PERIODONTAL SPECIFICATION OF CONTACT

The contact relation between teeth of an individual jaw is basic to the periodontal health.[14,15] There must be continuity of contact completely around the arch. Each tooth thus gives support to its neighbor on either side. This contact of tooth to tooth is also maintained by the transseptal periodontal membrane fibers acting as a connecting band, and by the roots of teeth embedded in the alveolar bone.

This contact relationship helps to maintain tooth stability and position in the arch in a horizontal plane and to hold functional movement within physiologic limits. Another function is to provide for the flow of the forces of occlusion around the arch through the contact area. Thus occlusal forces generated on alternate sides of the arch during mastication are supported by the whole arch as a unit. This flow of occlusal forces also provides important stimulation for the deep periodontal structures.

Anatomically, the contact area forms the top border of the embrasure space. Mechanically, it protects the embrasure contents from injury during mastication. Failure to restore the contact area results in periodontal disease that destroys the embrasure tissues and then attacks the deeper underlying periodontal structures.

ADDITIONAL PERIODONTAL SPECIFICATIONS

The periodontal specification "occlusion" in a restoration involves all of the occlusal relations of the teeth. It also involves all restorations from the simplest Class I to complicated bridge replacements.

Before new occlusal restorations are built, it is fundamental that the entire remaining dentition be balanced as a

13. Black, G. V. Operative dentistry, vol. 2. Chicago, Medico-Dental Pub. Co., 1936, p. 137.

14. Brodie, A. G. Significance of tooth form. Angle Orthodont. 4:335, 1934.

15. Bunting, R. W. Preventive dentistry. In American textbook of operative dentistry, ed. 8. Gabel, A. B., editor. Philadelphia, Lea & Febiger, 1947.

mastication unit in centric jaw relation and in the functional eccentric jaw positions.[9,11,12] Schuyler,[16,17] and McLean [9,18-20] have laid down the rules for balancing occlusion. Restorations and replacements are constructed so that they are in harmony with the balance already created in the remaining natural dentition. Failure to rebuild to a balanced occlusion results in compounding the forces of malocclusion and increasing the rate of destruction from periodontal disease. Attempting occlusal adjustment after restorations are made results in mutilation of the articulating anatomy, loss of metal protection of porcelain bridge pontics and often in complete destruction of the new restoration.

Occlusion and occlusal adjustment are the common denominator between periodontal and restorative service. Major centric and eccentric occlusal derangements should be corrected before treatment of advanced pockets. It is an important aid to successful healing as it eliminates the excessive mobility of teeth in premature contact. These teeth also tend to return to their original positions in the arch, often closing open contacts and improving the occluding contacting relationship. Adjustment of remaining minor discrepancies is done after complete periodontal healing, and again after all restorations are completed. A final "milling in" process simulating natural functional wear completes the adjustment program. It removes the small discrepancies remaining which were caused by the inherent limitations of the carbon marking technic.

Because of their extreme importance, diagnosis and an understanding of the status of occlusion, particularly centric occlusion, should be re-emphasized.[12,18,20,21] Occlusion can be spoken of as being "healthy" or "diseased," in the same manner as the periodontal tissues. Interdigitation and simultaneous tooth contact with the condyle resting passively in the fossa mean "healthy" centric occlusion. The lack of interdigitation and simultaneous tooth contact with the condyle resting passively in the fossa mean "diseased" centric occlusion. The Hickok retruder is a valuable and practical aid to quick diagnosis of centric jaw relation. The retruder insures a condyle resting passively in the fossa and closure on the hinge axis of the jaw.

The periodontal specifications of contour, margins and polish of a restoration have to do mainly with the proximal tooth surfaces.[1,14] They apply to a lesser degree to the other tooth surfaces as well. These proximal surfaces form the mesial and distal sides of the embrasure space and are in intimate physical contact with the periodontal tissues within this space. Therefore, these restored surfaces should reproduce the natural convexities of this portion of the tooth. They should have a high polish and the gingival margins should present no discrepancy or overhanging margins. Failure to restore the contour, polish and margins results in gross mechanical irritation to the embrasure tissues, and causes extensive periodontal disease.

The question of teeth with total bifurcation and trifurcation involvements is intimately connected with restorative procedures. Because of the importance of such involvements, careful clinical and roentgenographic diagnosis must be used.

In many instances, planning the reconstruction program is impossible until a decision is made as to what can be done with strategically located teeth so involved. Solutions to this problem include

16. Schuyler, C. Fundamental principles in the correction of occlusal disharmony. J.A.D.A. 22:1193 (July) 1935.

17. Schuyler, C. Correction of occlusal disharmony of the natural dentition. New York D. J. 13:445 (Oct.) 1947.

18. McLean, D. W. Physiologic vs. pathologic occlusion. J.A.D.A. & D. Cosmos 25:1593 (Oct.) 1938.

19. McLean, D. W. Pathologic occlusion: a major clinical problem. Fort. Rev. Chicago D. Soc. 8:6 (Dec. 15) 1944.

20. McLean, D. W. Diagnosis and correction of pathologic occlusion. J.A.D.A. 29:1202 (July) 1942.

21. Leonard, H. J. More about occlusal factor in periodontal disease. J. Periodont. 21:109 (April) 1950.

treatment of the area, devitalization of the tooth and resection of one root, splinting to adjacent teeth, and extraction.

THE INDIVIDUAL RESTORATION

The individual restoration is without doubt the most important of all the restorative procedures.[6,7] Its purpose is twofold: to restore parts lost by decay and so preserve the tooth as a functioning unit; and, secondly, to protect the integrity of the supporting structures by restoring the shape and form of the outer surface. These mechanical and periodontal specifications of a restoration have already been described.

Under healthy conditions, with periodic roentgenographic examination to discover decay, the individual restoration built to mechanical and periodontal specifications, plus excellent personal oral hygiene, will insure continued health of the dental machine indefinitely.

When periodontal disease is present, the individual restoration is an essential factor in treatment. Faulty restorations that fail to meet the tests of contact, contour, occlusion, margins and polish should be removed and replaced by properly designed restorations. More advanced chronic conditions with open contact spaces, tipped teeth and malpositioned teeth are corrected by building exaggerated contours, extensions on restorations for contact, and by redesigning the occlusal surface.

The advantages of the indirect gold inlay over the amalgam filling in satisfying the periodontal specifications of a restoration are obvious.[6,7] When occlusal reconstruction is required, gold inlays are the only possible solution to the problem. Thus the periodontal viewpoint immediately results in a great increase in the use of the gold inlay restoration.

THE FIXED BRIDGE

The fixed bridge in the replacement of missing teeth is the method of choice.[3,5,22] It fulfills the requirements of durability, function, comfort and limitation of movement impossible for the clasp removable bridge to achieve. From the periodontal viewpoint, it is axiomatic that any missing tooth from second molar to second molar should be replaced. Hirschfeld[8] has pointed out the degenerative sequela resulting from failure of replacement. As a rule, all replacements should be made fixed where possible, and removable appliances should be used only where it is impossible to construct a fixed bridge.

When there is normal bone support and unchanged tooth position, the fixed bridge restores the integrity of the arch and insures continued health of the mastication machine. Under these conditions, the method of construction using either complete fixation by soldering both ends of the pontic or the properly designed stress divider may be utilized.

When soldered at both ends, the whole bridge, including both abutments, must move together in function. This is contrary to individual physiologic tooth movement, and strains are generated which tend to dislodge the smaller abutment inlay. Thus a strong and more solid abutment casting must be built.

When the stress divider principle is used at one end it allows individual physiologic movement of each abutment. Thus no strains are generated. This allows the use of smaller and more conservative castings. Bodecker[23] pointed out that teeth move in function, but this physiologic movement is slight as the average width of the periodontal membrane is 0.20 to 0.30 mm. As Grubb[22] has pointed out,

> . . . in any restoration, fixed or removable, it would seem to be impossible to approach absolute fixation to the degree that the tissues

22. Grubb, H. D. Partial dentures with precision attachments. J.A.D.A. 42:154 (Feb.) 1951.

23. Bodecker, C. F. The physiological movement of teeth. D. Items Interest 58:129 (Feb.) 1936.

would be under stimulated. The problem is how to avoid excessive movement and overstimulation of the supporting structures rather than immobility.

Bartlett[3] favored the stress divider principle and outlined its advantages.

If periodontal disease has caused weakened bone support and tipped tooth position, the fixed bridge is essential. Its objective is to achieve as much fixation and support as possible and to prevent excessive movement. This is best accomplished by soldering both ends of the bridge where alignment permits. When there are unparalleled abutments, the stress divider joint has to be used. Although it is a second choice, it is still far superior to the clasp removable bridge. The splinting of two or more weakened teeth, rigidly soldered together to serve as a multiple bridge abutment, is a proved and standard technic to achieve stability. Other aids to weakened support are reduction in the size of the occlusal surface of both abutment and pontic tooth, reduction of vertical and horizontal masticating forces by optimum occlusal relation of supporting cusp and guiding inclines, and a ridge-to-ridge occluding contacting relationship instead of large flat surfaces. Thus, once again, the periodontal concept increases the necessity for fine inlay bridge work.

REMOVABLE BRIDGE AND PARTIAL DENTURE

The removable bridge and partial denture supplies replacement in instances of multiple loss of teeth and where replacement by fixed bridgework is impossible.[22] The purpose is to restore the integrity of the arch, distribute the forces of mastication over as many units as possible, and to preserve the remaining natural teeth.

Removable replacements fall into two classes: those retained by clasps fitted around the outside of the abutment teeth, and those retained by precision attachments fitted inside the circumference of the abutment teeth.

Dentists and patients alike who have had experience with both types of replacements agree that the precision attachment partial denture offers many advantages over the clasp partial denture. From the periodontal viewpoint these advantages are a rigidity similar to the fixed bridge, the splinting effect of solid bilateral construction, the occlusal load supported by both the tissues and abutment teeth[2] and the stimulation provided by intermittent pressure on the tissues.[4, 22, 24] Other advantages include mental and physical comfort for the patient, better function, better appearance and less caries. Also the normal tooth contours are maintained.[4, 22]

Under certain conditions, the clasp and the precision attachment can be used in combination on the same prosthesis. The clasp is used on the side where there is tooth support, and the attachment is used to best advantage on the side of the arch where there is tissue support.

The net effect is that the remaining teeth will last longer because of the precision attachment partial denture. With the average clasp partial denture, because of careless design and abuse, the opposite effect is produced and the loss of the remaining teeth is hastened. Some of the disadvantages of a poorly designed clasp partial denture which may cause disease include overloaded abutments, omission of, or inadequate, occlusal rests; continuous clasp tension, and palatal section impinging on the gingiva.[22, 4, 2]

When the clasp partial denture is properly designed, many of the disadvantages mentioned can be eliminated or minimized. Since necessity and economics will dictate the choice of the clasp partial denture in the majority of cases, more serious attention to the details of

24. Weaver, S. M. Precision attachments and their advantages in respect to underlying tissues. J.A.D.A. & D. Cosmos 25:1250 (Aug.) 1938.

good construction should be emphasized.

With normal bone support and a favorable bone factor,[25] years of satisfactory service may be expected from prostheses with precision attachment. Even an extreme distal extension prosthesis consisting of eight or more pontic teeth and two bilateral abutments attached to the cuspids or even to the lateral incisors may be very successful. Patients are most appreciative, particularly when these partial dentures are constructed for the lower jaw since they are spared the discomforts of the complete lower denture.

The greater the periodontal complications of bone loss with an unfavorable bone factor,[25] the greater the need for precision attachment partial dentures if the remaining teeth are to be retained. The use of the attachment principle in these instances is often the deciding factor between treatment and extraction of the remaining teeth. The double abutment principle, splinting of weak teeth, placing additional attachments in the lingual surfaces of fixed bridge pontics and in individual weak teeth, are procedures used in conjunction with the attachment partial denture in certain difficult cases.

The splinting procedure consists of tying together two or more teeth. The method of choice is a solder joint connecting a three-quarter crown gold casting. The most common application is where a weak tooth is splinted to a solid tooth. Whole groups of individually weak teeth also can be splinted together in this manner, and collectively they become solid and strong. This applies particularly to the lower anterior teeth. Such splinted tooth groups when used as attachment bridge abutments are further strengthened by the reciprocal support received from the appliance.

CONCLUSIONS

All types of mechanical restorations of lost teeth, when built to correct periodontal specifications, perform a biological function indispensable to the health of the periodontium. It is a biomechanical solution to dentistry's major problems—the saving of the teeth and maintaining their supporting structures in health and the treatment of a diseased mouth.

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25. Glickman, I. Experimental basis for the "bone factor" concept in periodontal disease. J. Periodont. 20:7 (Jan.) 1949.

Accurate Language • It is of the utmost consequence that strict accuracy should regulate our use of language, and that every one should acquire the power and the habit of expressing his thoughts with perspicuity and correctness. Few, indeed, can appreciate the real extent and importance of that influence which language has always exercised on human affairs, or can be aware how often these are determined by causes much slighter than are apparent to a superficial observer. False logic, disguised under specious phraseology, too often gains the assent of the unthinking multitude, disseminating far and wide the seeds of prejudice and error. Truisms pass current, and wear the semblance of profound wisdom, when dressed up in the tinsel garb of antithetical phrases, or set off by an imposing pomp of paradox. By a confused jargon of involved and mystical sentences, the imagination is easily inveigled into a transcendental region of clouds, and the understanding beguiled into the belief that it is acquiring knowledge and approaching truth. A misapplied or misapprehended term is sufficient to give rise to fierce and interminable disputes; a misnomer has turned the tide of popular opinion; a verbal sophism has decided a party question; an artful watchword, thrown among combustible materials, has kindled the flame of deadly warfare, and changed the destiny of an empire. *P. M. Roget, "Introduction to Roget's International Thesaurus of English Words and Phrases," revised ed., p. 11.*

Correction of abnormalities of occlusion

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Vigorous function of the teeth favors normal development and[1-7] maintenance of the periodontal tissues.[8,9,2,10] Wear of the teeth is a regular result of vigorous functional activities.[11,7] The changes which vigorous function and wear cause throughout the teeth and their supporting structures appear to be normal physiologic processes which are essential to maturation of both the deciduous and permanent dentitions.[2,11-15] Lack of function or indolent function of the teeth results in failure of normal development and normal maintenance of the periodontal tissues.[2,9,14,16] Function of the teeth also may cause injury to the periodontal tissues.[2,13,17,18] The damage in the periodontal tissues which arises from loading on the crowns of the teeth is almost invariably associated with tangential loading.[2,3,8,19,20] Poor resistance to injury is probably a common contributing factor in those instances where damage in the periodontal tissues results from functional activity of the teeth.[10,19-23] This paper will be confined to a discussion of those factors, conditions and technics which have a direct bearing on changing unfavorable function, or lack of function, so as to promote occlusal relations which will produce more favorable effects on the teeth and the periodontal tissues.

Two basic principles which have governed the development and application of the technics are (1) torque is the main injurious agent of traumatogenic occlusion, and (2) natural wear of the teeth is an ideal adjuster of occlusions.

Torque is defined in the Oxford English dictionary as "the twisting or rotary

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2. Box, H. K. Twelve periodontal studies. Toronto, University of Toronto Press, 1940, p. 42-55.

3. Kronfeld, Rudolf. Histopathology of the teeth and their surrounding structures, ed. 2. Philadelphia, Lea & Febiger, 1939.

4. Roger, A. P. Place of myofunctional treatment in the correction of malocclusion. D. Record 48:57, 1928.

5. Baker, L. W. A preliminary study of the influence of the forces of occlusion on the development of the bones of the skull. Int. J. Orthodont. & Oral Surg. 8:259, 1922.

6. Landsberger, R. Histological research concerning the growth of the alveolar process in its relation to the development of the tooth germ. D. Cosmos, 66:1334 (Dec.) 1924.

7. Waugh, L. M. Influence of diet on the jaws and face of the American Eskimo. J.A.D.A. & D. Cosmos 24:1640 (Oct.) 1937.

8. Williams, C. H. M. Present status of knowledge concerning the etiology of periodontal disease. Oral Surg., Oral Med. & Oral Path. 2:729 (June) 1949.

9. Williams, C. H. M., and Watt, D. G. Effects of physical consistency of food on the periodontium of the rat. National Research Council of Canada. Proceedings 13, Appendix 3.

10. Stillman, P. R., and McCall, J. O. A textbook of clinical periodontia, ed. 2. New York, Macmillan Co., 1937, p. 125-127.

11. Campbell, T. D. Dentition and palate of the Australian aboriginal. Adelaide, Hassell Press, 1925.

12. Jenkins, D. H. Maturation of the permanent dentition. Queensland D. J. 3:62 (Feb.) 1951.

13. Gottlieb, B., and Orban, B. Biology and pathology of the tooth and its supporting mechanism. New York, Macmillan Co., 1938.

14. Coolidge, E. D., and Hine, M. K. Periodontia, clinical pathology and treatment of the periodontal tissues. Philadelphia, Lea & Febiger, 1951.

15. Weinmann, J. P., and Sicher, H. Correlation of active and passive eruption. Bur 46:128 (Nov.) 1946.

16. King, J. D., and Gimson, A. P. Experimental investigations of parodontal disease in the ferret and related lesions in man. Brit. D. J. 82:126 (Sept. 19) 1947.

17. Goldman, H. M. Periodontia; a study of the histology, physiology and pathology of the periodontium and the treatment of its diseases, ed. 2. St. Louis, C. V. Mosby Co., 1949.

18. Stones, H. H. An experimental investigation into the association of traumatic occlusion with parodontal disease. Proc. Roy. Soc. Med. 31:479 (March) 1938.

force in a piece of mechanism" and rotation is defined as "the action of moving round a center." The term rotation is not used in this paper with the restricted connotation peculiar to its application in orthodontics. Orthodontists employ the word to indicate movement of a tooth around the long axis.[24] In this paper any movement of a tooth about a rotation center or axis will be considered rotation. Thus if a tangential loading causes the displacement of the crown of a tooth toward the labial side and of the apex of the root toward the lingual side, the tooth will be considered to have rotated about its rotation center. The rotation center usually is situated slightly apically to the mid-length of the root.

Rotation of a tooth within its socket usually results in areas of extreme compression, extreme tension and relative inactivity within the pericementum.[2, 3, 13, 14] The most obvious injuries usually have been demonstrated in the areas of extreme compression. Since torque and rotation, resulting from tangential loading of teeth, seem to be so important, it is desirable to understand the factors that promote or favor torque action and the conditions which resist torque. Technical procedures for control and correction of abnormal occlusal functions should be related to that understanding.

FACTORS PROMOTING TORQUE ACTIVITY

The following factors promote or favor torque activity:

Inclination of Contacting Occlusal Surfaces • This seems to be the most important single factor. Differences of 10 degrees, 15 degrees, and 20 degrees in the inclination of the functioning surface make striking changes in the torque potential. Pronounced overbite and high cusps usually are associated with steep inclinations while with short overbites and low cusps the inclination of the incisal and occlusal surfaces often is nearly horizontal.

Amount of Force • The significance of this factor probably has been greatly overstressed. It is controlled by the amount of muscular pressure exerted by the muscles of mastication, the number of teeth in simultaneous contact and the sensory reaction of the periodontal membrane.

Length of the Lever Arm • This is the distance from the point where the force is applied on the crown to the center of rotation in the root. It is divisible into the extraalveolar and intraalveolar portions. Long extraalveolar and short intraalveolar lever arms favor torque activity while short extraalveolar and long intraalveolar lever arms result in small torque potential.

Frequency of the Function • If all other factors are equal, a pair of teeth which function unfavorably and which function frequently will receive a greater torque influence than a pair which seldom come in contact. The arrangement of the teeth and the extent and direction of the movements of the mandible control the frequency with which pairs of teeth function.

RESISTANCE TO TORQUE

Resistance to torque and rotation is principally controlled by the following factors:

Size and Shape of Roots • A study performed in our laboratory has revealed

19. Orban, B. Traumatic occlusion and gum inflammation, J. Periodont. 10:39 (Jan.) 1939.

20. Gratzinger, M. Muscular force, occlusion and function as etiologic factors in periodontal disease. J. Periodont. 22:131 (July) 1951.

21. Becks, H. Systemic background of paradentosis. J.A.D.A. 28:1447 (Sept.) 1941.

22. Glickman, I. The experimental basis for the "bone factor" concept in periodontal disease. J. Periodont. 20:7 (Jan.) 1949.

23. Granger, E. R. Bio-mechanics of periodontal disease. J. Periodont. 21:98 (April) 1950.

24. Dorland, W. A. N. American illustrated medical dictionary, Philadelphia, W. B. Saunders Co., 1937.

that great variations of area of the roots of teeth exist. A random sample of 40 cuspid teeth revealed that the largest root had 325 square millimeters of surface, while the two smallest had 50 square millimeters of surface.[25] A classification of root forms which has proved useful for clinical practice is: long rectangular, long triangular, short rectangular and short triangular.

Amount of Supporting Structure • There seems to be little room for doubt that loss of supporting structure as a result of periodontitis simplex is often the cause of transforming the effect of functional loading of the teeth from physiologic to traumatogenic. If a root were strictly rectangular in form, loss of a third in length of the principal fiber attachment, from the amelocemental junction toward the apex, would result in a third reduction of the support of the tooth, whereas a similar loss of a third in length of attachment on a root of the same length, but of average taper, would result in approximately a 50 per cent reduction of the area to which the periodontal membrane is attached.

Thickness and Density of Periodontal Membrane • Density and thickness of the periodontal membrane will vary under the influence of different functional stimuli but they also may be affected by general systemic conditions.[21,22] A periodontal membrane of normal width and density will support the root against displacement more effectively than a wider and relatively fibropenic membrane.

OTHER MODIFYING FACTORS

There are certain factors and conditions which neither directly activate rotation nor directly resist it but which do modify the potential damage which might arise from certain occlusal disharmonies.

Direction of Movement of Mandible • In the majority of instances the chin-point, or the lower incisor teeth, move anterolaterally in relation to centric position during lateral functional excursions. But among a significant percentage of patients with natural teeth and pronounced overbites the chin-point may be observed to move straight laterally or even posterolaterally. It is important to determine the direction of movement of the mandible when attempting the development of a treatment plan. It is also important to recognize that only the most complex articulator machines will allow reproduction of straight lateral or posterolateral movements. The use of most articulators for study of correction of such instances will almost surely lead to inaccuracies.

Resistance of Periodontal Tissues to Injury • This factor often is difficult to assess but is probably of great importance. The history, the physician's report, and the speed and amount of the damage which has occurred in the periodontal tissues are employed as means of determining resistance to injury.

Natural Wear of the Teeth • This factor will be discussed later in the paper.

Difference of Inclination of the Anterior Teeth • If both upper and lower incisor teeth are in vertical alignment and there is a vertical overbite but no horizontal overbite, the incisor teeth will be in constant contact during both lateral and protrusive functional excursions. Furthermore, the direction of the loading on the incisors will be at right angles to their long axes. With the same vertical overbite but with a difference in the direction of the long axes of the incisors, such as exists when they both incline a little

25. Williams, C. H. M., and Brown, R. A method of measuring root area. Soon to be published in J. Canadian D. A.

anteriorly, the incisors probaby will be free of loading during lateral functions and during part of the protrusive excursions. The direction of the loading on the teeth also would be less unfavorable.

Steepness of the Cusps of Bicuspids and Molar • If the functioning planes of incisor and cuspid teeth are steep, then steep cusps on the posterior teeth are more favorable to a coordinated function than low cusps. The ideal, however, seems to be no overbite and low cusps.

Curve of Spee and Compensating Curve • As the steepness of the incisor and cuspid functioning plane increases it is desirable that the arc of the curve of Spee and of the compensating curve be shorter.

WEAR OF THE TEETH

Studies of the skulls of human beings of earlier times and of contemporary groups have indicated that vigorous function and wear of the teeth are an invariable accompaniment of well developed and well sustained dentitions. The relationship is probably cause and effect. The following steps in the process of wear may be regarded as physiologic:

1. The evolution of the incisal and occlusal surfaces follows a typical pattern. At first the bicuspids and molars are worn mainly at or near the tips of the cusps. Thus the raised portions of the occlusal surfaces are obviously reduced while areas which support centric occlusion are slightly reduced in vertical height. As cuspal inclines approach horizontal direction, exposed dentin cups out which causes the establishment of inverted-cusp patterns. This process usually is found to be most advanced on those teeth which erupted first and least advanced on teeth which are last in order of eruption.[11]

Vertical overbite of the incisor teeth of young persons usually is small if the dentitions are well developed.[7] Wear reduces overlap of the front teeth and allows the establishment of end-to-end relation in early adulthood.[7, 11] Exposed dentin of the incisors also cups out and small inverted cusp patterns are established.

The opinion that worn teeth are mechanically inefficient may be incorrect. But even if the relatively horizontal occlusal surfaces and inverted cusps do mean that increased muscular power is needed in mastication, there is evidence that an increased load in an apical direction may be tolerated by the periodontal tissues when an increased tangential load would not be tolerated.[2]

2. Wear of the teeth tends to favor distribution of the occlusal load over maximum numbers of tooth surfaces.

3. Vigorous function and wear of the teeth appear to favor continuous or cyclical appositional deposition of bone at the alveolar edge.[26] Frequently in well worn dentitions the distance from the amelocemental junction to the alveolar edge is not significantly greater than it was when the teeth were unworn.

4. After the cusps of the teeth have been nearly eliminated by wear, and the teeth have assumed an end-to-end relation, the areas of the teeth which support centric occlusion wear obviously. Since the facial proportion does not seem to be disturbed by wear of the teeth,[27] it must be that the teeth move toward the occlusal plane, not out of but accompanied by the supporting structures. Under the influence of this process the extraalveolar lever arm of the tooth constantly becomes shorter in relation to the intraalveolar arm. Of course, the deciduous dentition, in the latter part of its period of service, suffers shortening of the intraalveolar lever arm by resorption.

26. Weinmann, J. P. Bone changes related to eruption of teeth. Angle Orthodont. 11:83 (April) 1941.

27. Herzberg, F., and Holic, R. Anthropologic study of face height. Am. J. Orthodont. & Oral Surg. 29:90 (Feb.) 1943.

There is no intention of suggesting that artificial wear should be employed to imitate the degree of the changes just described. It will be suggested that conservative grinding may favor the operation of part of this process.

CLASSIFICATION OF OCCLUSAL RELATIONS

Benefit or damage to the periodontal tissues as a result of the occlusal function of the teeth are the result of the dynamic relations of the teeth. It is, therefore, desirable that a classification of occlusal relations should imply the probable effects of these dynamic relations. If classification is to be based on a single criterion, the overbite relation seems to serve best. The use of the classification in determining choice of method for correcting occlusions has served well in practice. The classification of overbite is as follows:

1. Normal overbite, 0 to 1.5 millimeters.

If any overbite exists, it seems necessary that certain modifying and compensating conditions exist. These conditions are natural wear of the teeth, difference in inclination of the incisors, anteroposterior incline planes of cuspids, bicuspids and molars, the curve of Spee and compensating curve.

2. Slightly abnormal overbite, 0 to 2 millimeters, not compensated by the factors named in 1.

In most of these cases normal natural wear, if it occurred, would bring about good to excellent occlusal relations in all ranges of function. In the absence of natural wear, artificial wear, or grinding, finds its ideal application.

3. Medium abnormal overbite, 2 to 4 millimeters, not compensated by the factors named in 1.

In most of these cases artificial wear of the teeth will allow the production of good lateral functional relations, but only fair incisal relations. Ideally the gross part of the malrelation should be corrected by orthodontic or, occasionally, prosthetic appliances, and the fine adjustment should be accomplished by grinding.

4. Decidedly abnormal overbite, 4 to 6 or more millimeters, not compensated by the factors named in 1.

Artificial wear of the teeth usually will accomplish a small to fair improvement for lateral functions and little or no improvement in incisal functions. The gross abnormalities of relationship should be corrected by orthodontic or, occasionally, by prosthetic devices and the fine adjustment should be attained by grinding.

It is encouraging that orthodontic treatment now is being successfully applied to patients 30 years of age and more. Decrease of overbite seems to be the most important single improvement that could be accomplished for many of the adult patients.

TREATMENT OF ABNORMAL OCCLUSAL RELATIONS

Immobilization of teeth by splinting procedures, using either fixed or removable devices, is a valuable means of treating or counteracting the undesirable effects of abnormal occlusal relations.[17,28] It seems to be indicated where loosened teeth would be supported by firm teeth or where splinting two or more teeth would enable them to support an uncommon loading. Splinting of teeth around the angles of the arches seems particularly efficacious and it certainly does not appear to be contraindicated.

Reduction of crowding of incisors often may be accomplished by extraction of a single tooth and by moving the adjoining teeth, by simple methods, to fill the space.[29] Anterior teeth which have

28. Miller, S. C. Oral diagnosis and treatment, ed. 2. Philadelphia, Blakiston Co., 1946.

29. Smith, R. Periodontal conditions associated with buckling of the lower anterior teeth: etiology, diagnosis and treatment. J.A.D.A. 34:303 (March 1) 1947.

migrated as a result of periodontal disease frequently will return spontaneously to more normal positions following successful treatment.[30] Simple appliances also may be used in such instances to bring the migrated teeth toward more favorable functional relations.

Although occlusal correction by grinding only occasionally is the ideal method, clinical experience indicates that it will produce some improvement in most of the relatively unworn dentitions of human beings today. Indication for its application may be determined by having the patient exhibit, at request, existing and true centric position (the existing centric position may be normal), right lateral position, left lateral position and protrusive position. Correction by grinding of centric position will be discussed later in the paper. To establish the eccentric positions, the patient is required to move the mandible until upper and lower cuspids are end-to-end at the lateral positions and until the incisors are end-to-end for the protrusive position. While the patient holds his jaw steadily at each of these eccentric positions, the answers for the following two questions are sought in turn.

If the patient had had more natural wear of the teeth would the occlusal relations at this eccentric position and the function between this eccentric position and centric position be better than they are?

Is it feasible to imitate the effects that natural wear would have produced?

If the answer to both these questions is "Yes," occlusal correction by grinding seems indicated. If the answer to either of them is "No" it is considered to be contraindicated. It is important to stress again that increased distribution of the loading over more teeth than were originally in contact, although significant, does not seem to be the most important requirement for improvement. It is often possible to reduce the torque potential significantly by reducing the inclination of contacting surfaces from 30 degrees to about 15 degrees without attaining any increase in the balance or distribution.

A two-phase technic for occlusal correction by artificial wearing has been developed. It has been in daily use in practice for many years and it seems efficient.

CORRECTION OF CENTRIC OCCLUSION

A large percentage of patients have an abnormal centric occlusion. However, only a minority of these cases may be efficiently corrected by grinding.

Orthodontic or prosthetic procedures, or occasionally extraction of teeth, may be used in the correction of abnormal centric occlusion.

If centric occlusion is to be corrected by grinding it receives attention before the eccentric positions are adjusted. Then each eccentric position and function is corrected by a two-phase technic which will be described.

There are a number of methods of inducing a patient to close his jaws in true centric occlusion. If the patient will swallow and allow the teeth to close until the first point of occlusal contact is established, the teeth usually will be extremely close to their true centric occlusion. If moderate grinding will change the number of and the inclination of the contacting surfaces of the teeth sufficiently so that the true centric occlusion will be assumed naturally and maintained, the procedure is indicated.

The patient should practice several times closing his jaws in true centric position using the method described. Carbon paper then is placed over all occlusal surfaces and when the teeth have reached the first point of contact they are tapped

30. Hirschfeld, I. Dynamic relationship between pathologically migrating teeth and inflammatory tissue in periodontal pockets. A clinical study. J. Periodont. 4:35 (Jan.) 1933.

lightly against the paper. Most of the grinding is done on the markings at or near the tips of the cusps rather than in the sulci with which they may interlock. The process is repeated until multiple contacting surfaces have been established. The patient then should automatically place his jaws in true centric position when he is asked to close his back teeth. When the teeth are closed at the corrected centric position, heavy biting pressure should not cause the teeth to slide toward the original centric position.

CORRECTING ECCENTRIC POSITIONS AND FUNCTIONS

Two observations which commonly may be made on unworn and slightly worn dentitions determine the technical procedures for correcting eccentric positions and functions by grinding. These observations are:

The areas of the cusps which contact when the jaws are in centric position do not contact in eccentric positions.

The surfaces of the cusps which contact during one eccentric function do not contact during other excursive movements of the mandible.

The technic for occlusal correction by artificial wear consists of two main phases for each eccentric function. In the first, the principal changes of size and form of the cusps are made quickly and to approximately what is expected to be the final patterns. In the second phase the fine coordination of moving relations of cusps is achieved. Both phases of correction are completed for each eccentric function before proceeding with the next. This system seems to contribute to an orderly efficient technic.

First Phase • The objective is to establish maximum distribution for the occlusal load and, at the same time, to alter the inclination of the cuspal surfaces to what will be approximately the final pattern.

The patient is trained to close his jaws on request directly to an eccentric position.

The order in which right and left lateral positions are corrected does not seem to have an important bearing on the final result.

It is usual to correct first the eccentric position and function which will mean the greatest amount of change in size and shape of the cusps or the incisal edges.

To assist the patient in assuming a lateral position of the jaws, a finger is pressed against the buccal surface of an upper second bicuspid and the patient is asked to close the lower teeth against the finger tip. Adjustment is designed to allow a wide range of lateral functional movements because observations on well-arranged dentitions, showing at least moderate wear, indicate that the buccal surfaces of the lower teeth will move laterally even beyond the buccal surfaces of the upper teeth when certain foods are being consumed.

With the teeth in eccentric position, many of the patients will exhibit individual contacts between relatively unworn upper and lower cuspid teeth. If the separations between other cusp points at this position range between zero and two millimeters, increased distribution of the loading usually can be attained by grinding. Such a condition as this will be used as a hypothetical case for discussion of the technic of artificial wearing. Carbon paper markings are obtained by having the patient close his teeth directly to the eccentric position to be corrected first—in this case a lateral position. The teeth are pressed tightly against the carbon paper, but without any obvious sliding or chewing motion. Thus, carbon paper marks will be produced on only those two surfaces which are immediately concerned. During the first phase of adjustment, all markings are obtained in this manner at the eccentric position.

The main bulk, but not all, of the grinding in the first phase is performed on the buccal cusps of the upper teeth and the lingual cusps of the lower teeth, provided the usual interdigitation exists. Thus, in this hypothetical case, the first main grinding would be on the upper cuspid and in such a direction as to establish approximately the incline plane which would have resulted if the tooth had worn naturally. The functioning plane extends from the point of contact at the limit of the lateral excursion to the point of contact at centric position. As the tooth wears naturally, or is worn artificially, the labioincisal angle of the wear facet moves apically while the centric support remains essentially constant. A line drawn from what will be the new position of the labioincisal angle to what is and will continue to be the centric occlusion marking would indicate the new pattern of the functioning surface. In the first phase of adjustment the grinding is stopped a little short of such a pattern. The same rule is applied to all cusps which may, as a result of grinding, come into contact during this first phase of adjustment.

In some cases occlusal contacts either exist at the beginning, or become established during the first phase of adjustment, on the opposite or balancing side of the arches when the mandible is in this eccentric position. If the operator believes that grinding should be only on the buccal cusps of the upper teeth and the lingual cusps of the lower teeth, he would, of course, not proceed. Contacts on the balancing side are almost always between the lingual cusps of the upper tooth and the buccal cusps of the lower tooth. Provided the carbon paper markings are produced with the teeth in lateral position, the areas of the cusps that are marked will be close to, but will not actually be, the areas that support centric occlusion. They may be ground by a restrained technic only on the areas marked by the carbon paper.

When the operator has established what seems to be the maximum distribution of the loading which it is feasible to attain by moderate change of tooth form, and when the ground surfaces favor smooth exercise of that particular eccentric function, the first phase of adjustment is completed. The second phase of adjustment for the same lateral function usually is performed in immediate succession.

Second Phase • The objective is to obtain the maximum of smooth coordination of the occlusal contacting surfaces during the movements from eccentric position to centric position.

With carbon paper over all incisal and occlusal surfaces, the patient is requested to close his jaws in the eccentric position for which the first phase adjustment has already been completed. The lower teeth then are caused to slide from the eccentric position to centric position. If the patient fails to accomplish eccentric to centric movement, he is allowed to move from centric to eccentric. One movement usually is sufficient to provide carbon paper markings and any more movements are likely to produce confusing evidence. All marked spots are ground with a fine stone and a light touch. The procedure is repeated until the sound of the functional movement, the tactile perception obtained by placing the fingers on the moving teeth, the carbon paper markings and the patient's report, indicate that the functional movement from the particular eccentric position to centric position is smooth and well coordinated. The adjustment for the other eccentric positions and functions then may be performed in the same manner.

Disturbance of existing centric occlusion by grinding is more likely to occur during the second phase of adjustment than during the first phase. The carbon paper markings which are established when the patient slides his teeth from

eccentric to centric positions, end where certain spot-like or line-like portions of the cusps support centric occlusion. With carbon paper between interdigitated teeth, the boundaries of the markings indicate the portions of the cusps which support centric occlusion. If fine grinding, therefore, is confined strictly to the areas showing the carbon paper markings, centric occlusion is unlikely to be disturbed. Clinical experience supports this opinion. If the incisor teeth are in contact at centric position to begin with, even slight closure of the bite at centric, as a result of adjustment of lateral functional movement, would be extremely noticeable to the patient. In those rare instances where the patient, with his jaws in centric occlusion, is conscious of an increased pressure on the incisor teeth, a delicate adjustment usually will suffice for correction.

At the first signs of sensitivity of tooth surfaces which are being ground, completion of the adjustment is postponed. The functioning relations are made as smooth as is feasible without exposing sensitive dentin surfaces and a period of at least six months usually is allowed to elapse. When sclerosis of dentin and the formation of secondary dentin have progressed sufficiently, additional grinding may be performed.

Teeth with weak supporting periodontal tissues or those with exceptionally heavy loading should be ground into, not out of, occlusion during the main adjustment procedures. This will insure that the occlusal patterns of such teeth will be suited to coordinated function with the other teeth, even if they are temporarily relieved of function and then come back into contact as they usually do.

Malaligned incisors are often strikingly improved in appearance by artificial wearing and in many cases certain shaping of such teeth can be accomplished without disturbance of the functioning surfaces. The appearance of the incisor teeth of many adults, which would not be corrected otherwise, can be improved by this simple method. When both phases of adjustment for incisal function are completed, carbon paper markings are left on the teeth to indicate the areas of the teeth important to function. All other areas may be shaped to obtain better appearance.

SUMMARY

Torque is the principal injurious agent of traumatogenic occlusion. Vigorous function and wear of the teeth are favorable to a series of coordinated changes which seem to constitute normal physiologic maturation of deciduous and permanent dentitions.

The more severe abnormalities of occlusion should be adjusted by orthodontic, prosthetic or even extraction procedures.

The more moderate abnormalities usually can be at least partially adjusted by artificial wearing of the teeth and, in a few instances, it is the ideal method for correction of occlusion.

The vigorous, comfortable and efficient function which the patient enjoys after adjustment of occlusal function is probably favorable to continued normal physiological processes of the teeth and their periodontal tissues.

Editor's Note • The paper entitled "The Role of Infection in Periodontal Disease," presented by Theodor Rosebury at the University of Michigan In-training Service Course on Periodontal Disease, September 10-15, 1951, was published in the April, 1952, issue of Oral Surgery, Oral Medicine and Oral Pathology, page 363.

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LON W. MORREY, D.D.S., EDITOR
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Editorials

Knowledge and uncertainty in the realm of periodontal disease

The series of papers on periodontal disease published in this number of THE JOURNAL, and the subsequent reports of evaluating committees to appear in the next issue, reflect professional knowledge and opinion as they exist today. The practices and views reported were developed mostly in the present century and owe very little to the past. To assert the modernity of this imposing body of information, however, is not to say that periodontal disease was unknown in ancient times or that its present problems and their solutions are altogether new in general outline.

The ancient Egyptians in their medical papyri took note of loose teeth and proposed remedies for them. Under the name *dentium commotio,* the disease was recognized in the medical works of the Middle Ages. A clear concept of the disease as a clinical entity was first set forth by Fauchard, the great French dentist, who in 1746 considered it a special kind of scurvy. His description, which is too long to quote here, presented most of the classic symptoms. From that day on, all practitioners who treat the mouth have been aware of the condition or conditions today called periodontal disease.

Many of the problems which in the early days arose to challenge the practitioner have persisted to the present. What are the causes of the disease? Is the condition single or manifold? Where is the essential pathosis situated? How may it best be dealt with? What may the practitioner expect as the outcome of his treatment? Each generation of clinicians and investigators has faced these questions; and though progress has been made in answering them, as the present extensive reports evidence, yet even today these interrogations have not been entirely answered to the satisfaction of all practitioners and scientists alike.

The most unsatisfactory aspects of the profession's knowledge of periodontal disease are of two sorts. They are either (1) contradictory views of clinical and laboratory workers, or (2) uncertainties due to the lack of evidence. Contradictory views, which are the more serious weaknesses, center largely around a few matters—such as the existence of periodontosis as a distinct entity, the relation of traumatic occlusion to pocket formation, and the regeneration and reattachment of tissues destroyed by disease. These disagreements and the parties to the controversies are well known in the profession, and consequently offer nothing new when they appear in the proceedings of the periodontal workshop. The uncertainties of periodontal knowledge, however, are not nearly so universally apprehended, and it is possible that some of them were uncovered for the first time by the deliberations of the evaluating committees. These gaps are important because future progress in diagnosis and treatment will depend largely on supplying the missing facts. These unsolved problems offer the profession numerous opportunities for specific research on essential details.

Some of these areas for further study may be taken at random from the statements of workshop participants. "The mode of attachment of the epithelium to the enamel is not yet a settled question." "A critical evaluation of the available reports shows that the significance of specific systemic factors as etiologic components in the pathogenesis of marginal periodontitis has not been established." "The mechanism of pocket formation, especially that of epithelial downgrowth, is still controversial." "It may be concluded that the concept of continuous passive eruption must be re-examined." "The psychosomatic factors constitute one area of investigation in which there is a particular need for studies under well-controlled conditions." "A survey of the available literature indicates that there have been no convincing studies reported to show that the physical character of the diet bears a proved relationship to the prevention of periodontal disease."

These are only a few of the doubts, reservations, and admissions of ignorance expressed by the participants in this conference of leading workers in the field of periodontal disease. Yet, in spite of the large negative element in the conclusions arrived at, there is an immense body of uncontroverted fact also presented. The proceedings of the workshop, individual papers and reports of evaluating committees, represent the combined contribution of nearly 200 specialists and research workers, and probably constitute the greatest aggregation and digest of the essential knowledge of periodontal conditions ever published in compact form.

United Coal Miners discontinue dental health service program

The United Mine Workers Welfare and Retirement Fund announced its discontinuance, on May 15, of all dental service for beneficiaries of the Fund's health program. This decision brought to a close an activity which had become increasingly disconcerting to the beneficiaries of the Fund and to the dental societies in many coal mining areas. A report of the action of the Fund officials is published on page 771 of this issue.

Undoubtedly the decision was influenced to a great extent by a conference held between representatives of the Association and Fund officials in Washington, D. C. on April 25. At that time a long look was directed at the program and a comprehensive discussion held on its weaknesses and potentialities. The dental representatives pointed out then that the American Dental Association's interest in the dental phase of the miners' health program is twofold: (1) to provide the beneficiaries with the highest type of dental service and at the same time fulfill the obligations of the Welfare and Retirement Fund, and (2) to aid in establishing more harmonious relations between officials of the Fund and members of the profession.

There is no question that the dental health of coal miners' families, like the dental health of families in other fields, calls for improvement, and credit is due those who have seen the need and tried to relieve it. However, many of the miners' dental service programs in the past, illy conceived and poorly executed, failed of their purpose—and failing left nothing but low quality dental service and ill will in their wake. Much of this ill will, as the representatives of the Association pointed out, could have been prevented had the leaders of the Fund and the profession been able to get together and reach agreement on the professional essentials of the project.

Apparently the officials of the Fund, in reevaluating the dental aspect of the miners' health program, have concluded that there is only one way out; since it is impossible to provide economically the quality of dental service which its beneficiaries should receive, it is best to discontinue the service—a very wise decision under the circumstances. However, since the objectives of the Fund are to provide health and welfare services it is inconceivable that Fund officials will fail to re-examine this problem with the object of developing a sound dental program for its members as dental health cannot be separated from general health.

Washington News Letter

As Congress goes into the closing weeks of its 1952 session, its working calendars disclose a fair amount of unfinished business of interest and importance to the dental profession. In subject matter, bills range from income tax relief to administrative strengthening of the Veterans Administration dental service. Which bills will be enacted into law—if any—cannot be foreseen; but it is certain that those measures failing to get through the legislative mill before adjournment will die with the 82nd Congress.

PENDING LEGISLATION

Reed-Keogh Bill • On May 13, the House Ways and Means Committee heard testimony by Paul E. Jones of Farmville, N.C., in support of the Reed-Keogh bill amending the tax laws to permit professional men to set aside yearly limited portions of their income for individual retirement programs.

Appearing as a member of the American Dental Association's Council on Legislation, Dr. Jones placed emphasis on the relatively few years of peak income enjoyed by dentists in buttressing his argument that a retirement plan of this kind is no more than equitable.

Even if every dentist in the country chose to participate in the Reed-Keogh plan for limited tax exemption, the revenue loss would be comparatively slight, said Dr. Jones. Even this loss would eventually be regained, he pointed out, when beneficiaries reached retirement age and began collecting their "old age pensions," which would be subject to income tax payments at the current rate.

Bonus Pay • Income is the subject of another pending Congressional bill, though the only dentists directly affected are those who are on duty in the Armed Forces, or who will enter military service in the future. At stake is the extension or termination of the $100 monthly special pay received by officers of the Medical and Dental Corps. Sen. Lester C. Hunt (D., Wyo.) leads the fight for continuation of this extra pay, which will expire September 1 unless Congress approves an extension.

Veterans Administration • The House Committee on Veterans Affairs is considering a bill recently introduced by Rep. J. Leonard Allen (D., La.) which would raise to a higher administrative level the dental division in the Veterans Administration's Department of Medicine and Surgery.

Although the opinions of Administrator Carl R. Gray, Jr., and Chief Medical Director Joel T. Boone have not yet been made known officially, they are expected to oppose the Allen bill on the ground that the dental service is virtually autonomous already. In this regard, however, there is a conspicuous divergence of opinion.

Women Dental Officers • Also in the "unfinished business" category is Senator Hunt's bill authorizing the commissioning of women dentists and physicians in the Armed Forces on the same basis as men. This bill passed the Senate without difficulty in March and would have passed the House early in May, but for an objection raised by Rep. Fred L. Crawford (R., Mich.). He was not opposed to the principle of commissioning women but to the fact that female osteopaths are unmentioned in the bill's provisions.

Outcome of the controversy is uncertain, but Capitol observers believe some sort of compromise will be worked out to permit enactment of the bill and thereby relieve, even if in a small way, the military's shortage of doctors.

VA Dental Program Budget • Only one pending Congressional bill, among those affecting dentistry, is certain to be enacted into law before the Senators and Representatives go home for the summer. This is the pending measure containing appropriations for medical, dental and hospital activities of Veterans Administration for the fiscal year beginning July 1. Here, the only question is how great the reductions will be when legislative action is completed and the bill is sent to the White House for signature.

Administrator Gray and Dr. Boone appeared before a Senate Appropriations Committee in May to appeal for restoration of budgetary funds that had previously been cut out by the House. The latter pointed out that if the House action were sustained, it would be necessary to authorize 15,600 fewer dental examinations and 24,700 fewer dental treatments than are being provided during the current fiscal year. The large majority of these examinations and treatments are performed by private dentists on a contractual basis. The VA wit-

nesses also explained that allotment of insufficient funds would interfere seriously with medical and dental residency programs.

There is little doubt that the Senate will be more lenient in dealing with the Veterans Administration budget and that compromise figures will be worked out when the bill goes into a joint House-Senate conference, probably early in June.

Delaney Report • Also expected some time during the month of June is publication of the Delaney Committee's report on fluoridation of community water supplies. This was the subject of extensive public hearings by the special investigating committee earlier this year. Although the report undoubtedly will make certain recommendations, these will have no binding effect of any kind.

UMW FUND DENTAL PROGRAM

Acting on short notice, the United Mine Workers Welfare and Retirement Fund eliminated all dental services for beneficiaries of the Fund's health program, effective as of May 15. The action came two weeks after a conference at Washington headquarters of U.M.W.A. between Warren F. Draper, executive medical officer, and a delegation from the American Dental Association. The delegation, led by Secretary Harold Hillenbrand, had recommended the addition of a dentist to the Fund's medical advisory committee, consultations with state dental societies and a number of proposals concerned with adherence to high professional standards.

Preceding his announcement that authorization of dental care was being abolished, Dr. Draper had informed Dr. Hillenbrand that if a dental program were continued, a dentist would be invited to become a member of the advisory committee.

FEDERAL TRADE COMMISSION

The Federal Trade Commission has issued an order citing Zonite Products, New York City, and Erwin, Wasey and Co., a New York advertising firm, for allegedly false advertising of Forhan's toothpaste. According to the Commission, the dentifrice "contains nothing the application of which is of therapeutic value in the prevention or cure of any disease or disorder of the teeth or gums. Its sole value is as a cleaning agent."

The respondents were ordered to stop representing that massaging the gums with Forhan's toothpaste is of any value in making them firmer or in prevention of periodontal diseases. According to the Commission's findings:

"Massage (as used by dentists) means a careful stroking or squeezing pressure applied to the gums in a particular area to achieve a specific purpose. Its proper use requires professional instruction. However, the preponderant weight of the qualified dental opinion is that massaging the gums in the sense of indiscriminately rubbing them is of no value and, in some cases, is actually harmful to the gums."

International Correspondence

NEWS FROM GREAT BRITAIN

Of present anxieties and future fears we have plenty these days in Britain, but as far as I can judge there is not one which is common to all dentists. In one way or the other, each of us is looking to the future, for in the days to come there are three events of outstanding significance due to take place.

THE COMING MONTHS

Some time during the summer, Parliament is due to enact two new laws which will have a profound effect on the practice of dentistry in Britain. The first will require patients receiving dental treatment other than dentures to pay the cost up to £1 ($2.79). Any amount over this sum will be claimed by the practitioner from the State after the patient has paid his contribution. If the treatment is £1 or less, the patient will pay the whole sum himself.

The second law is the Dentists Act, which will set up a new governing body, the General Dental Council, to replace the Dental Board of the United Kingdom. One of the clauses in this new act will require the General Dental Council to conduct an experiment in the use of dental ancillaries of a type similar to the New Zealand Dental Nurse.

The third event to which we are looking forward will take place July 18-26. Now that the International Dental Congress is only a few weeks ahead, interest is noticeably rising. Plans for the Congress are impressive.

It has recently been anounced that the Queen has agreed to become Patron of the Congress. This recognition is a great honour, and in Britain we appreciate and value this distinction which has been conferred upon the Congress.

We now know that it is to be the greatest dental meeting ever held in Britain, and it is to be on a scale exceeding anything we have known before in Europe. I have met a number of those active in the preparations for this outstanding event and from what they have told me of their work I am greatly impressed by the thoroughness of the arrangements. The Congress should run smoothly. There will be opportunities for all to see and learn the newest developments in scientific dentistry in all countries taking part in the meeting.

The scene of the Congress will be the Festival Hall built for the Festival of Britain which, it will be remembered, was held last year. In addition to the comfortable accommodation which the Hall offers, there will be available to foreign visitors simultaneous translations in the official languages of the Congress. This should help to speed up the proceedings during the discussions at the scientific meetings. Extensive accommodation is being reserved for the demonstrations and displays.

Most of the hospitals in London are preparing clinical meetings where television will be employed to demonstrate surgical operations. The experiences gained from the annual meeting of the British Dental Association held in London last year are proving of great value.

After the Congress, on July 28, a happy occasion for all interested in international dental affairs will take place in Edinburgh. Dr. Charles Nord, president of the Fédération Dentaire Internationale, will deliver the William Guy Memorial Lecture at the Hall of the Royal College of Surgeons. For all who remember this grand old man and for all in sympathy with the ideals which he held, it should be a memorable occasion.

NATIONAL HEALTH SERVICE REPORT

Dentures • The following figures, recently issued, show the number of full dentures supplied under the National Health Service:

July 1948-March 1950:	5,000,000
April 1950-March 1951:	3,780,000
April-December 1951:	2,216,000

The number of partial dentures supplied during the same periods:

July 1948-March 1950:	1,500,000
April 1950-March 1951:	1,150,000
April-December 1951:	780,000

Dental Operations • It has been estimated that during the last nine months of 1951 about 9,100,000 teeth were extracted, excluding those classified as emergency treatment, and 7,600,000 teeth were filled.

Treatments • The Minister of Health estimated that approximately 10,000,000 dental treatments or courses of treatment were given

in England and Wales. The estimated cost of orthodontic treatment during 1951 was about £500,000.

Estimates for 1952-53 • The gross payments to be made to dentists in the year April 1952 to March 1953 are estimated at £27,800,000 of which, under the scheme shortly to be introduced, the patients are to pay £9,850,000. This means the estimated cost to the State will be £17,950,000. In Scotland the corresponding figures are estimated as follows: gross payments to be made, £3,270,000; patients' contributions, £1,080,000, and cost to the State, £2,200,000.

These figures show the decline in dentists' earnings quite dramatically and are cited by those who are urging the Government to reconsider quickly the present scale of fees.

Included in the proposals for England is one to increase the number of Regional Dental Officers from 18 to 30. This will be welcome as it should increase the efficiency of the service by cutting down the delay which occurs when a practitioner's estimate has to be considered clinically by one of the Regional Dental Officers. Many practitioners believe that where there is an adequate number of RDO's the abuses for which the National Health Service has been criticised will cease.

THE SCHOOL DENTAL SERVICE

One effect of the reduced activity of practitioners in the Health Service which has followed the introduction of charges for dentures, is the reverse turn in the tide which has affected the School Dental Service. In a recent debate in the House of Commons, Mr. John Baird, who is a dental surgeon as well as an M.P., gave the present strength of the School Dental Service as below 900 dentists. In the last year, the service had a net increase of 40 dentists. This increase is a small figure, but it is a straw in the wind.

During the past four months, I have noticed this trend toward school dentistry. At least six of my former students have applied for posts in the School Dental Service, an occurrence unknown during the past four years.

With this trend toward school dentistry, the question naturally arises, will this continue to such an extent that the New Zealand type of nurse will be unnecessary?

As Mr. Baird pointed out, the figure for a good efficient service is about 3,000; but it must be borne in mind that all children do not accept the treatment that is required and advised. One school dentist told me that of 16 appointments made for one day, fewer than half turned up for treatment. I was a school dental officer for a year in 1937-38 and if 50 per cent of those who required treatment accepted it, I was pleased.

DR. HAROLD CHAPMAN HONORED

Recently, I drew attention to celebrations in honour of two of our oldest and most distinguished doyens, Dr. Lilian Lindsay and Sir Frank Colyer. It is with great pleasure I report that in April, Dr. Harold Chapman was entertained in the London Hospital at a luncheon marking his forty years of service on the staff of this famous school. He was presented with a silver salver.

It was my good fortune to have a few words with Dr. Chapman a month or two ago, and his many friends in the United States of America will be glad to know how well he was looking and how keenly he is interested in dentistry both here and across the Atlantic.

Dr. Chapman studied under Angle and received his D.D.S. from the University of Pennsylvania in 1904. He was the first practitioner in Britain to devote his practice entirely to orthodontics. He was a charter member of the British Society for the Study of Orthodontics. His overseas friends who attend the International Dental Congress will be pleased to find that, for the second time, he is president of the orthodontic society.

John Boyes, Newcastle-on-Tyne

Association Affairs

ERNEST G. SLOMAN, SPEAKER OF HOUSE OF DELEGATES, DIES

Ernest G. Sloman of San Francisco, dean of the College of Physicians and Surgeons and officer of the American Dental Association, died on April 30 at the age of 56.

Dr. Sloman was immediate past-president of the American Association of Dental Schools and was currently representing the dental profession on the President's Commission on the Health Needs of the Nation and the Commission on Financing of Hospital Care.

Ernest G. Sloman

An active member of the American Dental Association, Dr. Sloman was the current speaker of the House of Delegates, a former second vice-president, and past chairman of the Judicial Council. He was also a former member of the National Board of Dental Examiners and a member and former regent of the American College of Dentists.

Dr. Sloman held memberships in the International Association of Dental Research, the American Association of Dental Editors and the American Association for the Advancement of Science. In 1947, he was elected to the Supreme Council of Psi Omega and served as Grand Recorder of the fraternity.

A native of San Francisco, Dr. Sloman was graduated from the College of Physicians and Surgeons in 1921 and had served continuously since that time as a full-time member of the faculty, becoming dean of the school in 1938. At the time of his death, he was also professor of anatomy at the College of Physicians and Surgeons and lecturer in dental surgery at Stanford Medical School.

Dr. Sloman held offices and honorary memberships in local, state and regional societies, as well as in national organizations.

In 1947, as a tribute to his services to the dental profession, he was awarded the honorary degree of Doctor of Science from the University of Southern California.

He is survived by two daughters, a sister, and two brothers, one of whom is Victor E. Sloman, San Francisco dentist.

ASSOCIATION SPOKESMAN ENDORSES PENSION BILL

The Reed and Keogh bills, which provide for individual retirement programs, were strongly endorsed in the testimony of Paul E. Jones of Farmville, N. C., who represented the American Dental Association in recent hearings of the Committee on Ways and Means of the House of Representatives.

Dr. Jones contended that the bills, HR 4371 and 4373, would relieve dentists of an inequitable tax burden. His testimony in part stated, "The dentist almost invariably falls within the class of taxpayers who have a relatively limited number of high earning years. His extended period of education and training, plus the years spent in building up

his practice, combine to compress both his total earning years and his high earning years."

The Reed and Keogh bills would permit persons, especially the self-employed, to deduct from their taxable incomes any funds paid to a pension fund sponsored by an authorized organization, such as a dental society. The persons who finance their own pensions in this way would pay income taxes on their fund when they receive it back as income beginning at the age of 60.

Dr. Jones asserted that the immediate revenue loss to the government would not be more than $13,000,000 annually, even if every dentist availed himself of the benefits of the plan to the maximum extent. He summarized, "The American Dental Association in supporting HR 4371 and 4373 urges that Congress give the private practitioner the same opportunity given the corporate employee to provide voluntarily for his own future."

PUBLICATION SPECIALISTS TO ADDRESS DENTAL EDITORS

A complete morning session will be devoted to "Modernizing a Dental Journal —Publication Mechanics" at the conference of dental editors to be held June 23-24 in the Central Office. The session will be conducted by Kenneth B. Butler, president and general manager of The Wayside Press, Inc. of Mendota, Ill.

Other meetings during the conference will be devoted to finances, advertising, art and style. Dorothea Radusch, president of the American Association of Dental Editors, will discuss "The Regional Versus the State Journal"; Charles A. Wilkie, chairman of the Council on Journalism of the American Dental Association, will speak on "Functions and Purposes of a State Dental Society Journal." The organizations which the two represent are co-sponsors of the conference.

Among the speakers are William F. Schoen, editor of the *Illinois Dental Journal;* William L. Barnum, former editor of the Oregon State Dental Association's *Journal;* George B. Denton, chief manuscript editor of the American Dental Association, and George Likeness, executive director of the Butler Typo-Design Research Center of Mendota, Ill.

Participants in a special advertising session will be J. J. Hollister, business manager of the American Dental Association; Alfred J. Jackson, of the American Medical Association, and J. J. Nevin, vice-president of Coe Laboratories, Inc., Chicago.

REGISTRY OF ORAL PATHOLOGY TO RESTORE KODACHROME SETS

The Association's Registry of Oral Pathology is now withdrawing from circulation its kodachrome loan sets in order that they may be reconstituted and restored to their original value, it was announced May 12 by Col. Joseph L. Bernier, secretary.

The project of restoration will be completed in six weeks; meanwhile, requests will be placed on file and honored when the sets are again available, Col. Bernier stated. Requests should be addressed to the director, Armed Forces Institute of Pathology, Washington 25, D. C.

DEADLINE SET FOR APPLYING FOR LIFE MEMBERSHIPS

July 8 will be the last day on which the Association will accept 1952 applications for life memberships. The Board of Trustees and House of Delegates will act upon these applications at the annual session in September.

Any dentist who has reached the age of 65 and has been a member of the American Dental Association for 35 years, may apply for a life membership. A certificate is awarded to each life member and he is not required to pay annual dues in the Association as long

as he maintains active membership in his constituent and component societies.

Application forms may be obtained from the Membership Department, American Dental Association, 222 East Superior Street, Chicago 11.

CONCEPTS OF OCCLUSION TO BE TOPIC OF MEETING

Five commentators will participate in the discussion of "Concepts of Occlusion" at the Conference on Dental Nomenclature, June 20-21 in the Association's Central Office.

Harry Sicher of Chicago will comment on Albert A. Dahlberg's report, Thomas D. Speidel of Minneapolis on Touro M. Graber's report, Oswald M. Dresen of Milwaukee on Carl O. Boucher's report, Maynard K. Hine of Indianapolis on Paul E. Boyle's report and J. H. Johnson of Toronto on Orion H. Stuteville's report.

The conference, sponsored by the Bureau of Library and Indexing Service, will be conducted by George B. Denton, research consultant of the Association.

Further information may be obtained by writing to Donald A. Washburn, director of the Library and Indexing Service, 222 East Superior Street, Chicago 11.

URGE EMERGENCY MEDICAL TRAINING FOR DENTISTS

A recommendation that all dentists and dental students be trained in emergency medical care of casualties as soon as possible, was issued recently by the subcommittee on civil defense of the Association's Committee on National Emergency Dental Service.

This and other recommendations were formulated at the subcommittee's April 26 meeting and were presented for joint action at a subsequent meeting with the American Medical Association's Council on National Emergency Medical Service.

David E. Hunn of Troy, N. Y., chairman of the subcommittee, reported that 16 constituent dental societies have active civil defense units, 15 have made nominal appointments to such committees and at least 15 are not yet organized to participate in civil defense. These were the findings of a recent mail survey of the Association's constituent societies.

The subcommittee approved the civil defense program proposed for dentists in a new Federal Civil Defense Administration monograph by Russell W. Bunting, dental consultant to the FCDA. It was recommended that the text be published in a future issue of THE JOURNAL.

DENTISTS TOLD HOW TO MEET ANTI-FLUORIDATION CHARGES

Fluoridation facts of importance to the dentist and his community are contained in the Association's newest information bulletin, "Fluoridation of Water Supplies —A Job for the Local Community." Research findings and statistics are presented, along with suggestions for "Informing the Public" and "Meeting the Arguments."

The bulletin has been sent to all members of the American Dental Association. Additional copies may be obtained free by writing to the Association's Bureau of Public Information, 222 East Superior Street, Chicago 11.

DEMAND FOR PAMPHLET BRINGS PRICE DECREASE

The price of the Council on Dental Health's newest pamphlet, "Why Your Dentist Recommends Fluoridation," has been cut by 50 per cent, it was announced recently by the Council. As a result of an "overwhelming demand" for the pamphlet, an additional 200,000 copies have been ordered and a substantial reduction in cost has been obtained.

The pamphlet, designated as G2 by the Order Department, is designed for

distribution to lay groups and civic organizations. Prices are now: 90 cents for 100 copies, $3.40 for 500 copies and $5.90 for 1,000.

25 ST. LOUIS HOTELS OFFER HOUSING FOR ANNUAL SESSION

Good accommodations in St. Louis are still available for those who wish to register for the 93rd annual session of the American Dental Association, September 8-11. Henry F. Westhoff of St. Louis, chairman of the committee on local arrangements, has announced that 25 of the city's leading hotels are cooperating to see that all visitors to the meeting are adequately housed.

Scientific sessions of the 93rd annual session will be presented at Kiel Auditorium, within walking distance of the downtown hotels. A highlight of the scientific program will be the combined meeting of all 11 scientific sections for discussion of antibiotics and focal infection on Wednesday, September 10.

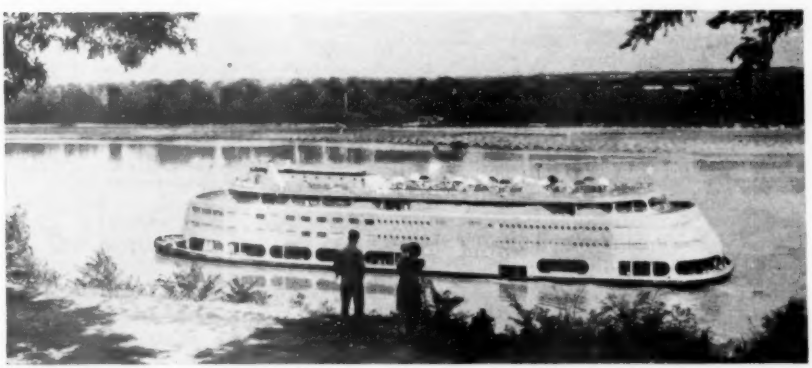
In addition, 60 essayists will participate in the separate sessions of the 11 scientific sections. Color television clinics will be staged in a specially constructed dental office and will be transmitted, by means of new technical improvements, in clear, illuminated pictures of dental operations. A continuous program of the latest scientific films will be presented, as well as 200 table clinics and 50 scientific exhibits.

Persons who plan to attend the annual session have been requested to fill out the housing application which appears on page A-35 of this issue. Advance reservations indicate a large attendance.

COUNCIL ON DENTAL EDUCATION APPROVES TWO PROGRAMS

The four-year programs of the Schools of Dentistry at Emory University and the University of Alabama were fully approved by the Association's Council on Dental Education at its meeting, May 15-16 in the Central Office.

The Council also heard a report by its Committee on the Training of Dental Hygienists, which is currently inspecting 25 schools of dental hygiene. The



The steamer, Admiral, shown sailing down the Mississippi River, is a modern counterpart of the colorful old St. Louis riverboats. Steamer trips on the Mississippi are among the colorful attractions of St. Louis, where the 93rd annual meeting of the American Dental Association will be held September 8-11. An entertainment and recreation program for dentists and their wives is being planned by the local committee on arrangements.

committee listed recommendations of general application which have grown out of the survey thus far. The recommendations will be sent to all dental hygiene schools.

The Council approved the factual report, soon to be released by the U.S. Public Health Service, on the financial survey of dental schools conducted jointly by the USPHS and the Council.

A further report on the Council's action on aptitude testing, internship and residency programs and dental specialties, will be published in the next issue of THE JOURNAL.

COUNCIL APPROVES 30 MORE HOSPITAL DENTAL DIVISIONS

At its recent meeting in the Central Office, the Council on Hospital Dental Service approved 30 hospitals which have dental departments meeting the basic standards of the Association. The Council acted upon the recommendations of hospital examining committees appointed by constituent dental societies.

The dental departments of more than 360 hospitals have been approved by the Council, according to Frank B. Hower of Louisville, Ky., chairman.

Dental Societies

DEDICATE NEW HEADQUARTERS OF SECOND DISTRICT SOCIETY

The new headquarters building of the Second District Dental Society of the State of New York was dedicated at ceremonies held May 23. The building, at 111 Ft. Greene Place, Brooklyn, is the first erected as a headquarters for a dental society in the United States.

Speakers at the midday ceremonies included C. Raymond Wells, past president of the American Dental Association, the president of the Borough of Brooklyn, civic health officials and dental society representatives.

The new building, a two-floor brick structure, houses a lecture hall, classroom, laboratory, model dental office and administrative offices. Built at a cost of $100,000, the headquarters has been financed by membership loan subscriptions and assessments. The Society is composed of 2,500 dentists from Kings and Richmond counties, New York.

MICHIGAN, OKLAHOMA DENTAL SOCIETIES NAME EXECUTIVES

The appointment of H. Leon Snow of Oklahoma City as executive secretary of the Michigan State Dental Association, was announced at the recent state meeting in Lansing, Mich. Mr. Snow is the former executive secretary of the Oklahoma State Dental Association and managing editor of the Oklahoma state *Journal*. He succeeds the late Henry C. Gerber, Jr., who was executive secretary of the Michigan society for 13 years.

William Howard of Oklahoma City is the new executive secretary of the Oklahoma State Dental Association, replacing Mr. Snow. Mr. Howard was the director of public relations for Oklahoma City University and previously served as executive vice-president and secretary of the Oklahoma City Junior Chamber of Commerce.

NEW ORLEANS DENTISTS TO CONFER NOVEMBER 9-12

The New Orleans Dental Association will hold its annual conference November 9-12 at the Roosevelt Hotel, New Orleans. J. Melville Smith, III, of New Orleans is chairman of the conference. Further information may be obtained by writing to M. R. Matta, Secretary, 629 Maison Blanche Building, New Orleans.

May 8, 1908

The regular monthly meeting of the El Paso County Odontological Society was held at the Acacia Hotel on this date.

The following members were present: Drs. Bartlett, Smith, Hayden, McKay, Baker, Linton, DeBerry, Wilson, Gray, Grannis, Johnston, Burton, Fleming and Nelson.

Minutes of the previous meeting were read. The President directed that same be corrected.

Considerable discussion was had led by Dr. McKay, with reference to the brown stain, or so-called "Colorado Stain" so frequently found upon children's teeth in Colorado Springs. A motion was made by Dr. Gray and seconded by Dr. Wilson that a suitable patient exhibiting this condition be secured and that this society pay the expenses of such patient to the State Dental Association meeting at Boulder, in June, so that the case might be inspected by members of that body, etc. Carried. Dr. Grannis moved that two of our members be appointed to look after this matter. Seconded by Dr. Gray. Carried. The President appointed Drs. Fleming and McKay on this committee

F. C. Jocelin
Secy

Reproduced above is a page from the original 1908 book of minutes of the Colorado Springs (Colo.) Dental Society, then known as the El Paso County Odontological Society.

On this page is recorded the earliest known organized action in the investigation of what later became known as "mottled enamel" and still later, after its cause had been determined, as dental fluorosis. THE JOURNAL is indebted to Frederick S. McKay of Colorado Springs, pioneer investigator referred to in the illustration, for a copy of this unique document in the history of dentistry.

Dental Education

TUFTS BERKSHIRE CONFERENCE TO BE CONDUCTED JUNE 15-19

The third annual Berkshire Conference in Periodontology and Oral Pathology will be conducted June 15-19 in Lenox, Mass., by the division of graduate and postgraduate studies, Tufts College Dental School.

The conference will consist of four days of lectures, seminars and panel discussions of clinical problems by teachers and clinicians in dentistry, medicine and related fields.

Further information regarding the conference may be obtained by writing to the Berkshire Conference, Tufts College Dental School, 136 Harrison Avenue, Boston 11.

LOUISVILLE TO OFFER DENTAL HYGIENE CURRICULUM

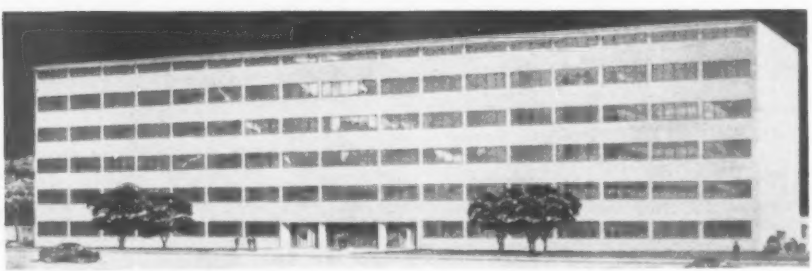
The University of Louisville dental school has opened a two-year curriculum leading to certification in dental hygiene, it was recently announced. The first class will enter September 22, 1952. Applications for admission to this class are now being accepted; inquiries may be addressed to the Office of the Dean, University of Louisville, School of Dentistry, 129 East Broadway, Louisville 2, Ky.

POSTGRADUATE COURSES OFFERED BY DENTAL SCHOOLS

Illinois • A two-year, full-time postgraduate and graduate course in the basic sciences, affording limited clinical experience, is being offered by the department of oral and maxillofacial surgery, University of Illinois. The course will carry credit toward a Master of Science degree.

Applicants now are being accepted for enrollment next September. Further information may be obtained from the College of Dentistry, University of Illinois, 808 South Wood Street, Chicago 12.

Pennsylvania • The University of Pennsylvania dental school will offer a postgraduate course for endodontics teachers, September 12-14. The first day will be devoted to a review of principles and psychology of learning and teaching. The



A new classroom and laboratory building under construction at the University of Illinois Professional Colleges, Chicago, is shown in an architect's sketch. The dental school, which will share the building's facilities with the colleges of medicine and pharmacy, will be enabled to increase its enrollment by 12 per cent when the building is completed late in 1953. Construction on the building was started on April 25.

second and third days will be concerned with the technics of teaching endodontics, together with consideration of demonstration lessons, the clinic and audio-visual aids in teaching.

Further information may be obtained from the Thomas W. Evans Museum and Dental Institute, University of Pennsylvania, 4001 Spruce Street, Philadelphia 4.

University of Washington • A course entitled "Dentistry for Children" will be offered June 17-20 at the University of Washington dental school, Seattle, by Kenneth A. Easlick, professor of dentistry, University of Michigan.

The course will include lectures, laboratory sessions, demonstrations and clinical sessions designed "to emphasize practical application of the most recent developments in operative technics, preventive procedures and pulp therapy." Dr. Easlick will be assisted by members of the pedodontics staff, University of Washington dental school. Enrollment will be limited to 15.

Application forms may be obtained by writing to the director of postgraduate dental education, School of Dentistry, University of Washington, Seattle 5.

National Defense

SEVEN PRIORITY I DENTISTS INDUCTED IN CALL-UP OF 335

Seven dentists who refused to apply for commissions were inducted into the Armed Forces as privates during the April call-up of 335 Priority I registrants by the Selective Service System. All but these seven applied for commissions, it was reported by Col. Richard H. Eanes, chief medical officer of the System.

Another 175 dentists were ordered to apply for commissions or face induction during May; reports indicate that no additional refusals have been encountered.

Priority I registrants are dentists who were trained at government expense or deferred from service to attend dental school during World War II and who have served less than 90 days.

As the pool of Priority I registrants is now exhausted, the services may soon call Priority II registrants and Reserves who have served fewer than 12 months.

It is estimated that in the next 13 months, the Armed Forces will need about 4,000 dental officers because of the expansion of the military services and the discharge of dental officers completing their tours of duty.

Public Health

UNITED MINE WORKERS FUND TERMINATES DENTAL PROGRAM

The United Mine Workers of America Welfare and Retirement Fund has discontinued its dental program. As of May 15, the Fund ceased to authorize dental services to its beneficiaries, it has been announced by Warren F. Draper, executive medical officer of the Fund.

The announcement was contained in a letter to Harold Hillenbrand, secretary of the American Dental Association, and followed a conference at which Fund officials and Association representatives discussed the complaints of constituent and component dental societies regarding the Fund's dental program in certain areas.

Dr. Draper's letter stated that, although the Fund would discontinue authorizing dental services, it "will collaborate to the fullest possible extent with the Public Health Service, the Children's Bureau, and State and local health departments in developing and extending their activities for the betterment of dental health."

FORT WORTH, TEXAS, ADOPTS FLUORIDATION

On May 7, the city council of Fort Worth, Texas, voted to begin the fluoridation of the city's water supply. Fort Worth has a population of 227,000 and is the fourth largest city in Texas. Although there was some opposition to the measure, it received strong support from the local medical and dental societies.

NEW ENGLAND HEALTH MEETING TO DISCUSS DENTAL PROBLEMS

Orthodontic problems in preschool children will be discussed by Wilbur D. Johnston of New Haven, Conn., and present dental health needs of children will be outlined by Irving W. Eichenbaum of New Britain, Conn., in a forum on dental and school health programs at the New England Health Institute June 18-20 at the University of Connecticut, Storrs, Conn.

The viewpoints of a dentist, a sanitary engineer and a biochemist regarding fluoridation will also be presented at the Institute, during a panel discussion of the measure. Participating in the discussion will be Frank E. Law, dentist of Boston; Charles R. Cox, sanitary engineer, and Harold C. Hodge, biochemist.

The Institute is sponsored by federal, state and private health agencies in six New England states. Further information may be obtained from the Connecticut State Department of Health, 165 Capitol Ave., Hartford 15.

Legislation

SENATE COMMITTEE APPROVES BILL TO EXTEND BONUS PAY

The Senate Armed Services Committee has reported favorably on the Hunt bill, S 3019, to extend a $100-a-month bonus pay to physicians and dentists who enter the federal services before July 1, 1953.

Present legislation authorizing the incentive pay will expire September 1. Dentists now in the Armed Forces and the U. S. Public Health Service will continue to receive the monthly bonus, but dentists entering the services after September 1 will be ineligible for the payments unless new legislation is passed.

If the Hunt bill is enacted, all dentists now in federal service, and all who enter until July 1, 1953, will be eligible for the bonus pay for a period of 30 years, for an aggregate payment not to exceed $36,000 each.

The Senate committee also approved an amendment which would extend the bonus payments to those retired medical and dental officers who are recalled to active duty. They are the only group of medical and dental officers on active duty who do not receive the additional pay.

BILL PROPOSES CHANGES IN VA DENTAL DIVISION

A bill to give the chief dental officer of the Veterans Administration greater authority in administering the VA dental program, is now being considered by the Veterans' Affairs Committee of the House of Representatives.

The legislation would also create the post of Chief Dental Director of the Dental Service in the VA Department of Medicine and Surgery. At the present time, the chief dental officer's title is Assistant Chief Medical Director for Dental Services.

The bill, HR 7507, provides that the Chief Dental Director be given the nec-

essary authority and responsibility for the standards in the dental program and for inspections, training programs, personnel, research and budget.

Rep. J. L. Allen (D., La.) introduced the bill on April 22.

WISCONSIN TO TEST DENTAL APPLICANTS IN SCIENCES

On July 1 a new Wisconsin law will go into effect, requiring dentists to pass a preliminary examination in the basic sciences before becoming eligible to take the dental board examination. Heretofore, dental applicants for state licenses were exempt from this basic science test, although other practitioners in the healing arts were required to pass it.

The preliminary examination will be administered by a separate board composed of persons not connected with medicine or dentistry. Rhode Island is the only other state with this requirement for dental applicants.

BILL SEEKS FEDERAL LOANS FOR NEEDY COLLEGE STUDENTS

A bill providing for federal loans to enable borrowers to obtain advanced education, was introduced into the Senate on April 28 by Sen. William Langer (R., N.D.). The bill, S 3077, would authorize the Commissioner of Education to lend a maximum of $1,000 to each approved applicant desiring education beyond the high school level, including professional education.

To finance the loans, the Langer bill proposes an appropriation of fifty million dollars, which would constitute a revolving fund from which loans would be made and to which they would be repaid.

The bill has been referred to the Committee on Labor and Public Welfare.

International

CANADIAN DENTISTS PLAN GOLDEN JUBILEE MEETING

Seven general assemblies, as well as table and group clinics, will be presented at the Golden Jubilee meeting of the Canadian Dental Association, to be held June 15-18 in Vancouver, British Columbia.

The seven general assemblies will feature lectures by George Hollenback of Los Angeles on operative dentistry, Philip Jay of Ann Arbor on preventive dentistry, Earl Pound of Los Angeles on prosthetic dentistry, Saul Robinson of Portland, Ore., on prosthetic dentistry (progressive group clinic), John Kuratli of Portland on inlays, crown and bridgework, H. M. Worth of Toronto on diagnostic roentgenology, and Roy West of Seattle on oral surgery for the general practitioner.

Among the dentists who will present group clinics are seven from the United States: G. D. Stibbs of Seattle, D. H. Hagen of Seattle, D. H. Gylenberg of Long View, Wash., B. Jankelson of Seattle, J. Ingle of Seattle, A. A. Schultz of Seattle and F. L. Jacobson of Seattle.

DENTAL RESEARCHERS TO MEET AT INTERNATIONAL CONGRESS

A section has been organized for dental research workers to discuss their mutual problems at the eleventh International Dental Congress, to be held July 19-26 in London.

According to the preliminary program of the Congress, the scientific sessions will be conducted from July 21 through July 25, composed of 20 international reports, three symposiums and 100 selected dem-

onstrations. The reports, which have been published in the *International Dental Journal,* will be presented in summary and will be discussed by 40 official openers.

United States dental authorities who will deliver reports are Isaac Schour of Chicago, Malcolm Carr of New York, Stanley D. Tylman of Chicago, Robert G. Kesel of Chicago, L. I. Grossman of Philadelphia, Shailer Peterson of Chicago and Balint Orban of Chicago.

Discussion openers will include J. P. Weinmann of Chicago, Harold Hillenbrand of Chicago, J. A. Salzmann of New York, Ernest R. Granger of Mt. Vernon, N. Y., John H. Sillman of New York, E. W. Skinner of Chicago, Harry Sicher of Chicago, C. Marshall-Day of Boston and John H. Knutson of Washington, D. C.

ITALIAN DENTAL ASSOCIATION TO MEET IN OCTOBER

The twenty-seventh Congress of the Italian Dental Association will meet with the first International Triennial of Prosthetic Dentistry in Rome during the second half of October, 1952.

During the meeting of the Italian Dental Association (A.M.D.I.) four topics will be discussed: pedodontics, physiology of mastication in relation to prosthetics, lesions of the soft tissues of the mouth, and analgesia and anesthesia.

Conferees at the prosthetic dentistry meeting will show materials and models currently being used. A special exposition will show the advancement of dental prosthetics through the centuries. American dentists who may have historic dental materials, equipment, instruments or models have been invited to show them during the Congress.

Further information may be obtained from Andrea K. Ovadia, Viale Regina Margherita N. 287 b, Rome, Italy. Dr. Ovadia is secretary-general of the combined Congress.

DENTAL LEADERS OF INDIA ASK AMERICAN COOPERATION

The dental health week committee of Calcutta, India, has requested the cooperation of the United States dental organizations in the planning and conduct of this year's dental health week exhibition. Mahomed Hansa, a member of the committee, requests "advice as well as posters, leaflets and other publicity materials."

Dr. Hansa, who is president of the Calcutta Dental College and Hospital, has also addressed an inquiry to dental schools and societies requesting an exchange of United States publications with his school's publication, *Dentindia.* "The hard currency shortage over here prevents us from ordering as much dental literature as we wish we could," Dr. Hansa adds.

Both publications and materials for dental health week may be sent to Mahomed Hansa, Calcutta Dental College and Hospital, P. O. Box 16006, Calcutta 17, India.

U. S. DENTISTS TO ADDRESS EUROPEAN ORTHODONTISTS

The twenty-ninth Congress of the European Orthodontic Society, to meet July 14-17 at The Hague, Netherlands, will number among its reporters Allan G. Brodie of Chicago, Andrew F. Jackson of Philadelphia, C. F. A. Morrees of Boston. Saul M. Bien of New York will present a clinic.

BRITISH HOUSE OF LORDS DEBATES DENTISTS BILL

In Great Britain's House of Lords, the Dentists Bill reached the report stage in April, after major debate on proposals to train and employ a class of ancillary dental workers to fill and extract the teeth of school children.

The Bill, which would amend the Den-

tists Act of 1921, proposes the following: (1) the establishment of an independent General Dental Council as the governing body of dentistry; (2) the registration of foreign applicants who are judged to be competent dentists by the General Dental Council; (3) the establishment of a three-year experiment in which ancillary dental workers would be trained to fill and extract teeth and would be employed in the school service.

The last provision has been the subject of controversy. The only amendment which has been written into the bill on this matter provides that the training of ancillaries must not "materially impair the facilities for the training of dental students existing at the passing of the Act."

General

OPEN NATIONAL HEADQUARTERS OF N.A.D.L. IN WASHINGTON

The National Association of Dental Laboratories has established headquarters at the Dupont Circle Building, 1346 Connecticut Avenue, N.W., Washington 6, D.C., it was announced recently by Paul A. Slone, executive secretary.

PLAN MEETING OF AMERICAN ACADEMY OF PERIODONTOLOGY

A three-day meeting of the American Academy of Periodontology will convene September 4 at the Statler Hotel, St. Louis. Further information on the program may be obtained from the organization's secretary, Clarke E. Chamberlain, 115 North Street, Peoria 5, Ill.

SUPREME COURT UPHOLDS OREGON MEDICAL SOCIETY

The United States Supreme Court recently declared that the Oregon State Medical Society had not violated anti-trust laws when it instituted its own prepaid medical care plan. The Court upheld the Society in an anti-trust suit brought by the Justice Department, which charged that the Society monopolized the business of prepaid medical care in Oregon.

GREAT LAKES SOCIETY OF ORAL SURGEONS TO MEET

The Great Lakes Society of Oral Surgeons, composed of members in Ohio, Michigan and Western Pennsylvania, will meet September 21 at the Statler Hotel, Cleveland. The meeting will extend from 9 A.M. to 4 P.M., according to P. J. Aufderheide of Cleveland, president.

UNIVERSITY OF PENNSYLVANIA DENTAL ALUMNI TO MEET

An award of merit will be presented to a dental leader by the Dental Alumni Society of the University of Pennsylvania dental school at its annual meeting June 20. Paul E. Bomberger of Lancaster, Pa., president of the Society, will preside at the organization's annual meeting and its annual dinner.

On June 19, members of the reunion classes will present a full day of refresher courses composed of symposiums on dental materials and technics, oral surgery, periodontics and oral cancer. Further information may be obtained by writing Z. T. Jackaway, 1506 East Susquehanna Avenue, Philadelphia 25.

ORAL PATHOLOGY BOARD ANNOUNCES EXAMINATION

The American Board of Oral Pathology will conduct its next certification examination September 7 in St. Louis. Applicants who wish to have their credentials evaluated in anticipation of appearing before the examining committee are requested to write promptly to Col. Joseph

L. Bernier, secretary, American Board of Oral Pathology, Armed Forces Institute of Pathology, Seventh Street and Independence Avenue, S.W., Washington, D. C.

Application blanks may also be procured from Colonel Bernier's office.

ORAL SURGERY BOARD SCHEDULES EXAMINATIONS

Examination for certification by the American Board of Oral Surgery will be held October 5-8 at the Jung Hotel, New Orleans. Further information may be obtained by writing to Leslie M. Fitz-Gerald, secretary, 718 Roshek Building, Dubuque, Iowa.

J. W. KEMPER AND G. W. MOORE OF MICHIGAN FACULTY DIE

Two eminent faculty members of the University of Michigan dental school died recently; they were John W. Kemper and George R. Moore.

John W. Kemper, head of the dental school's department of oral surgery, died May 8 at the age of 60. A physician as well as a dentist, he had been head of the oral surgery department since 1935 and had served as a faculty member continuously since 1929.

A native of Indiana, Dr. Kemper was graduated from the University of Michigan dental school in 1917. After practicing dentistry for six years, he enrolled at the University's medical school and was graduated in 1927. Although he practiced as a resident physician at the University Hospital, he returned to dentistry as an oral surgeon in 1929, when he joined the faculty of the dental school.

Dr. Kemper was a member of the American Dental Association, American Medical Association, American College of Surgeons, American College of Dentists, American Board of Plastic Surgeons, Omicron Kappa Upsilon and Phi Rho Sigma. In 1947-48, he served as president of the American College of Maxillo-Facial Surgery and in 1939 he was a member of the National Committee on Hospital Dental Services of the American College of Dentists.

Surviving are his widow and a sister.

George R. Moore, professor of dentistry (orthodontics) at the University of Michigan, died April 6 at the age of 52. He was at St. Petersburg, Fla., at the time of his death.

A member of the University's faculty since 1924, Dr. Moore was internationally-known as an orthodontist, lecturing in the Netherlands, England, Mexico and South America. He was president-elect of the American Association of Orthodontics, a former president of the Great Lakes Society of Orthodontists and former chairman of the section on orthodontics of the American Dental Association. He was a fellow of the American College of Dentists and held memberships in the International Association of Dental Research, American Association for the Advancement of Science, Omicron Kappa Upsilon and the Fédération Dentaire Internationale.

Dr. Moore is survived by his widow, two daughters and a son.

DENTAL EXAMINERS' SEMINAR TO DISCUSS TEST TECHNICS

State examiners and school representatives will discuss licensure examinations in operative dentistry, oral diagnosis and treatment planning, and prosthodontics during the seminar of the American Association of Dental Examiners, to be held September 5-6 in St. Louis. Lester M. Goodell, attorney for the Kansas Dental Board, will address the luncheon meeting September 5.

Further information and a program of the seminar workshop may be obtained by writing to Carl A. Bumstead, secretary-treasurer, 924 Stuart Building, Lincoln, Neb.

DIRECTOR OF NEW HOSPITAL ACCREDITING AGENCY NAMED

Edwin L. Crosby, president-elect of the American Hospital Association and director of the Johns Hopkins Hospital, Baltimore, has accepted the full time position of director of the newly-organized Joint Commission on the Accreditation of Hospitals.

The Commission will be operated jointly by the American Hospital Association, American Medical Association, American College of Surgeons, American College of Physicians and the Canadian Medical Association. The Commission will assume the hospital standardization activities carried on by the American College of Surgeons for the past 34 years.

Dr. Crosby, a physician and hospital administrator, will assume his new duties on September 1, with headquarters in Chicago.

EQUIPMENT BUSINESS SOLD, LINE OF GOLDS RETAINED

The Dee line of dental equipment and merchandise has been sold by Handy and Harman of Chicago and Los Angeles, to the Kerr Manufacturing Company of Detroit. Handy and Harman will continue to manufacture their line of dental golds.

NEW YORK ACADEMY OF DENTISTRY MEETS

Henry Hicks of Greenwich, Conn., was installed as president of the New York Academy of Dentistry at its annual meeting recently. He succeeds Lowrie J. Porter of New York. Other officers for the coming year are: Ralph J. Bowman of New York, president-elect; Roland E. Morse of Ridgewood, N. J., vice-president; William Ward Tracy of New York, secretary, and Gustav P. Frahm of Flushing, treasurer.

The New York Academy of Dentistry was founded in 1921 and is composed of four hundred dentists engaged in research, teaching or practice, who have been chosen for membership as a result of their contributions to the advancement of the profession.

JOHN N. CROUSE FUND GRANTS $15,000 TO DENTAL RESEARCH

The John N. Crouse Endowment Fund, administered by the Dental Protective Association of the United States, has awarded $5,000 to each of three dental research projects. The recipients are the dental schools of the University of Illinois, Northwestern University and Loyola University of Chicago.

The Dental Protective Association, founded 50 years ago to fight process patents, functioned until 1915, when the American Dental Association took up that work. The $50,000 in the organization's treasury at that time was placed in trust in 1938, under the name of the John N. Crouse Endowment Fund. The interest may be allowed to accumulate, or the interest plus two per cent of the principal may be used each year for research, fellowships or professional activities. This is the first year in which the fund has been used. Grants are paid by the fund trustees upon the recommendation of the Protective Association's professional committee.

J. B. CALDWELL, KURT THOMA TO DISCUSS ORAL SURGERY

A report on maxillofacial surgery will be presented by Col. J. B. Caldwell of the Army Dental Corps and will be discussed by Kurt H. Thoma on June 23, during the military-civilian "Symposium on Military Medicine" at Murphy Army Hospital, Waltham, Mass. Colonel Caldwell is chief of the oral surgery section at

Walter Reed Army Hospital, Washington, D.C.

Dentists in the area may attend this and other lectures, to be presented June 23-25, by writing or phoning the hospital for an invitation and program.

PHOTOGRAPHER TO DESCRIBE CLEFT PALATE PHOTOGRAPHY

The role of color photography in corrective work with cleft palate children will be described by Frederick T. Sharp of Springfield, Ill., during the August 12 meeting of the Photographic Society of America, at the Hotel New Yorker, New York.

Mr. Sharp, clinical photographer of the Division of Services for Crippled Children at the University of Illinois, will participate in an all-day program on photography in medicine and biology. Dentists in the area are invited. Further information may be obtained from the Photographic Society of America, 2005 Walnut Street, Philadelphia 3.

COLOMBIAN DEAN DESCRIBES SEVEN-YEAR DENTAL PROGRAM

Leonel Estrada, orthodontist and dean of the College of Dentistry, Medellin, Colombia, is now on a tour of dental schools in the United States, sponsored by a grant from the Kellogg Foundation.

Dr. Estrada attended the recent meeting of the American Association of Dental Schools and continued eastward on a six-weeks tour. He is a graduate of the College of Dentistry at Medellin and took his graduate training in orthodontics at Columbia University, New York. Dr. Estrada is accompanied by Javier Echavarria, professor of oral diagnosis at Medellin and president of the F.O.C. (Colombian Odontological Federation), who is now studying in the United States under a Kellogg Foundation grant.

Dr. Estrada listed the lack of teachers as the major problem in South American dental schools. He pointed out that the dental graduate in Colombia spends one year in pre-dental study, five years in dental school and one year as a Public Health dentist in a town which has no dentist. The Medellin school is supported by equal subsidies from the Colombian government and the state.

"I am grateful to the deans and faculty members in the United States for their sincerity and consideration," Dr. Estrada said. "We have much to learn and are exceedingly happy to be given an opportunity to visit your schools."

NEW YORK INSTITUTE PLANS WINTER TERM

The New York Institute of Clinical Oral Pathology has announced its courses for the October-May term. A 16-session course in clinical oral pathology will be presented on two evenings each month. An eight-session course in diagnostic and surgical technics will be presented one evening each month. Both courses are approved by the Veterans Administration and the University of the State of New York.

Information may be obtained from Theodor Blum, Secretary, 101 East 79th Street, New York 21.

DENTAL SEMINAR SCHEDULED IN EIGHT TENNESSEE CENTERS

The Tennessee State Dental Association, in cooperation with the Tennessee Department of Public Health, has scheduled its next seminar program, to be presented successively in eight district centers, for the period June 16-July 11.

The subjects of the 13-hour seminar program will be "Dental Materials," presented by Eugene Skinner of Northwestern University Dental School and "Dental Therapeutics with Special Reference to Root Canal Therapy," presented by F. D. Ostrander of the University of Michigan School of Dentistry. The pro-

gram is to be conducted in each district center either during the morning and the afternoon of two days or during the afternoon of three days.

PHYSICIAN TO DISCUSS NEW DENTAL ANESTHETICS

The value of thiamylal sodium and new muscle relaxants in dental anesthesia will be discussed by Peere C. Lund of Johnstown, Pa., at the fall meeting of the American Society for the Advancement of General Anesthesia in Dentistry, October 27 in New York.

Dr. Lund, a physician, is a diplomate of the American Board of Anesthesiology and director of anesthesia at the Conemaugh Valley Memorial Hospital, Johnstown, Pa.

At the same meeting, M. Hillel Feldman, director of dental service at the Lincoln Hospital, New York, will discuss the use of inhalation anesthesia for the cardiac patient. Further information may be obtained from Dr. Feldman, 730 Fifth Avenue, New York 19.

COMMISSION TO STUDY COST OF HOSPITAL CARE

The Commission on Financing of Hospital Care launched its national study on April 19, when members agreed on the organization and methods of its research program. The two-year fact-finding program will study four basic problems: the current financial position of hospitals; varying patterns of medical practice in relation to hospital care; financing of hospital care for the nonwage, low income, rural and chronic illness groups, and problems associated with voluntary prepayment of hospital care.

Organized independently last November, the Commission is composed of 34 members financed by $500,000 in grants from foundations. The late Ernest G. Sloman was the only dental representative on the Commission.

WISCONSIN DENTAL STUDY CLUB TO MEET AUGUST 14-16

The Wisconsin Dental Study Club will hold its annual meeting August 14-16 at the Loraine Hotel, Madison, Wis. Further information may be obtained by writing to R. H. Moore, secretary, 227 North Street, Madison.

PARENTS' MAGAZINE CARRIES ARTICLE ON FLUORIDATION

In the May issue of *Parents' Magazine* is an article on fluoridation of domestic water supplies entitled "The Magic of Fluorine." In addition to reviewing the history of fluoridation and reporting on the controlled studies of this measure, the article provides information for civic leaders on how to go about getting the benefits of the measure for their community.

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All expressions of opinions and all statements of supposed facts are published on the authority of the writer over whose signature they appear, and are not to be regarded as expressing the views of the American Dental Association, unless such statements or opinions have been adopted by the Association. *Articles* are accepted with the understanding that they have not been published previously and that they are submitted solely to THE JOURNAL. *Advertising copy* must conform to the official standards established by the American Dental Association. *Notice of change of address* should be received one month before the change is to be effective. *Orders for reprints of* JOURNAL articles must be received by the Business Manager's office not later than the 15th of the month of issue in order to secure advantage of the lowest possible prices. Orders received after this date are subject to special quotation.

Announcements

MEETINGS OF THE AMERICAN DENTAL ASSOCIATION

Ninety-Third Annual Session	Sept. 8-11, 1952	St. Louis
Ninety-Fourth Annual Session	Sept. 28-Oct. 1, 1953	Cleveland
Ninety-Fifth Annual Session	Nov. 8-11, 1954	Miami

MEETINGS OF STATE SOCIETIES

State	Date	Place	Secretary	Address
Alabama			F. A. Finney, Jr.,	1045 Forrest Ave., Gadsden
Alaska	July 14-17	Anchorage	R. A. Smithson	Box 1293, Anchorage
Arizona			E. M. Scott	17 E. Welden Ave., Phoenix
Arkansas	Oct. 26-28	Little Rock	D. M. Hamm	White Bldg., Clarksville
California	Apr. 13-15, 1953	San Francisco	L. R. Ludwigsen	450 Sutter St., San Francisco
S. California			C. A. Moss	1401 S. Hope St., Los Angeles
Colorado	Oct. 5-8	Colorado Springs	R. A. Downs	1422 Grant St., Denver
Connecticut			E. S. Arnold	37 Linnard Rd., W. Hartford
Delaware	Jan. 14, 1953	Wilmington	J. J. Ficca	2130 Lancaster Ave., Wilmington
District of Columbia			K. H. Wood	202-1835 Eye St., N. W. Washington
Florida	Apr. 19-22, 1953	St. Petersburg	C. T. Brown	602 Citizens Bldg., Tampa 2
Georgia	Oct. 12-15	Savannah	J. M. Heard, Jr.	803 Persons Bldg., Macon
Hawaii	June 11-14	Honolulu	J. H. Dawe	810 N. Vineyard St., Honolulu 17
Idaho	June 30-July 2	Sun Valley	F. E. Carpenter	123 Main Ave., W. Twin Falls
Illinois			P. W. Clopper	623 Jefferson Bldg., Peoria 2
Indiana			E. E. Ewbank	Kingman
Iowa			H. I. Wilson	639 Insurance Exchange Bldg., Des Moines
Kansas			F. A. Richmond	861 Brotherhood Bldg., Kansas City
Kentucky	Apr. 7-9, 1953	Louisville	A. B. Coxwell	1976 Douglas Blvd., Louisville
Louisiana	Apr. 29-May 2, 1953	New Orleans	J. S. Bernhard	2515 Line Ave., Shreveport
Maine	June	Rockland	S. Gower	Skowhegan
Maryland			E. L. Pessagno, Jr.	415 Medical Arts Bldg., Baltimore 1
Massachusetts			H. E. Tingley	12 Bay State Road, Boston
Michigan			F. Wertheimer	Michigan Dept. of Health, Lansing
Minnesota	Feb. 23-25, 1953	Minneapolis	C. V. E. Cassel	242 Lowry Medical Arts Bldg., St. Paul
Mississippi	June 9-12	Biloxi	H. M. Campbell	102 Professional Bldg., Tupelo
Missouri			R. R. Rhoades	201 Merchants Bank Bldg., Jefferson City
Montana			C. S. Renouard	304 Phoenix Bldg., Butte
Nebraska			F. A. Pierson	1112 Federal Securities Bldg., Lincoln
Nevada	June	Reno	R. La Fond	Medical Dental Bldg., Reno
New Hampshire	June 29-July 1	Bretton Woods	F. E. Williams	814 Elm St., Manchester
New Jersey	April 22-24, 1953	Atlantic City	J. G. Carr	407 Cooper St., Camden
New Mexico			J. S. Eilar	Suite 7, Medical Arts Square, Albuquerque
New York			C. A. Wilkie	1 Hanson Place, Brooklyn
North Carolina			B. N. Walker	Liberty Life Bldg., Charlotte
North Dakota			J. H. Lunday	302 Black Bldg., Fargo
Ohio			E. G. Jones	185 E. State St., Columbus
Oklahoma			R. C. Calkin	107½ W. Oklahoma, Guthrie
Oregon	Mar. 2-4, 1953	Portland	H. C. Fixott, Jr.	308 Selling Bldg., Portland
Panama Canal Zone	Dec. 9		F. D. Buckley	Box 293, Rodman
Pennsylvania			M. D. Zimmerman	217 State St., Harrisburg
Puerto Rico	Jan. 1953		A. Quiñones	San German
Rhode Island			E. L. Bessette	1745 Broad St., Cranston 5
South Carolina		Greenville	J. R. Owings	7 Medical Court, Greenville
South Dakota			E. W. Elmen	Sioux Falls
Tennessee			W. L. McCulley	Brown Bldg., Cleveland
Texas	May 10-14, 1953	Houston	W. Ogle	3607 Gaston Ave., Medical Bldg., Dallas

State	Date	Place	Secretary	Address
Utah	June 11-14	Zion National Park	E. O. Thompson	915 Medical Arts Bldg., Salt Lake City
Vermont			W. J. Reardon	Rutland
Virginia			J. E. John	804 Medical Arts Bldg., Roanoke
Washington			C. R. Flood	230 Medical-Dental Bldg., Seattle
West Virginia	July 14-16	White Sulphur Springs	A. B. Drake	508 West Virginia Bldg., Huntington 1
Wisconsin	Apr. 6-9, 1953	Milwaukee	R. A. Mason	964 N. 27th St., Milwaukee
Wyoming		Casper	T. J. Drew	State Capitol Bldg., Cheyenne

MEETINGS OF STATE BOARDS OF DENTAL EXAMINERS

State	Date	Place	Secretary	Address
Alabama	June 16	Birmingham	M. D. Edwards	132 Adams Ave., Montgomery
Alaska	July 7-11	Anchorage	J. H. Geyer	P. O. Box 915, Juneau
Arizona	Aug. 11-16	Florence	R. K. Trueblood	25 N. Second Ave., Glendale
Arkansas	June 23-26	Little Rock	H. O. Weatherly	205 Halter Bldg., Conway
California	June 23	Los Angeles	K. I. Nesbitt	507 Polk St., San Francisco 2
	June 28	San Francisco		
Colorado	June 23-27	Denver	C. K. Phillips	724 Republic Bldg., Denver 2
Connecticut	June 16-21*	Bridgeport	C. G. Brooks	302 State St., New London
Delaware	June 16-18*	Wilmington	P. K. Musselm	143 W. Main St., Newark
District of Columbia	June 2-6	Washington	W. M. Falls	1801 Eye St., N. W. Washington 6
Florida	July 7-12*	Jacksonville	A. W. Kellner	P. O. Box 155, Hollywood
Georgia			S. H. Yarbrough	Medical Arts Bldg., Columbus
Hawaii	Aug. 27-Sept. 1*	Honolulu	H. L. Houvener	427 Dillingham Bldg., Honolulu
Idaho	June 23	Boise	W. K. Adams	1624 Arlington, Caldwell
Illinois	June 24-27	Chicago	W. A. McKee	503 Wood Bldg., Benton
	Oct. 21-24			
Indiana	June 17-20*	Indianapolis	C. A. Frech	504 Broadway, Gary
Iowa			H. M. Willits	719 Roshek Bldg., Dubuque
Kansas	June 2-5	Kansas City, Mo.	G. L. Teall	Box 71, Hiawatha
Kentucky	June 2-5	Louisville	R. I. Todd	Western Union Bldg., Richmond
Louisiana	June 1-7	New Orleans	A. R. de Nux	Marksville
Maine	June 16-19	Boston	R. A. Derbyshire	Box 387, Skowhegan
Maryland	June 9-11	Baltimore	H. Levin	3429 Park Heights Ave., Baltimore 15
Massachusetts	June 23, 24, 26, 27‡	Boston	R. A. Barrette	Rm. 33 State House, Boston
	June 25-27‡‡			
Michigan	June 9-13	Ann Arbor	J. L. Champagne	3714 W. McNichols Road, Detroit 21
Minnesota	June 16-20	Minneapolis	F. A. Larson	1632 Washington St., N. E., Minneapolis
Mississippi	June 17-19*	Jackson	J. C. Boswell	508 Lamar Life Bldg., Jackson
Missouri	June 9, 10, 13, 14	St. Louis	R. R. Rhoades	414 Central Trust Bldg., Jefferson City
	June 11-14	Kansas City		
Montana	July 14-17	Helena	V. J. Quinn	Box 865, Helena
Nebraska	June 3-7	Lincoln	H. E. Weber	1210 Federal Securities Bldg., Lincoln
Nevada	June 2	Reno	G. C. Steinmiller	Suite 6, Masonic Temple, P. O. Box 2473, Reno
New Hampshire	June 20, 21	Boston	D. J. Freese	4 Wall St., Concord
New Jersey	June 2, 3**‡‡	Philadelphia	W. A. Wilson	150 E. State St., Trenton 8
	June 4-6§	Trenton		
	June 12, 13**	New York		
New Mexico	June 23-26	Santa Fe	J. J. Clarke, Sr.	Artesia
New York	June 19, 20**	New York	D. W. Beier	23 S. Pearl St., Albany 7
	June 24-27§			
North Carolina	June 23-26	Raleigh	F. O. Alford	1109 Liberty Life Bldg., Charlotte 2
North Dakota	July 7-11	Fargo	A. B. Crabtree	Ellendale
Ohio	June 23-27	Columbus	H. B. Smith	336 E. State St., Columbus
Oklahoma	June 19-21	Oklahoma City	J. B. Cole	706 Asp St., Norman
Oregon	June 9-12*	Portland	F. L. Utter	506 Pioneer Trust Bldg., Salem
Pennsylvania	June 19-21*	Philadelphia	A. M. Stinson	Dept. of Public Instruction, Box 911, Harrisburg
	June 21††			
	June 23-25†	Philadelphia and Pittsburgh		
Puerto Rico	Sept.	Santurce	L. C. Coll	Box 9156, Santurce
Rhode Island	June 16-18	Providence	F. M. Hackett	267 Academy Ave., Providence
South Carolina	June 26-28	Columbia	T. C. Sparks	1315 Pickens St., Columbia
South Dakota	June 22-25	Sioux Falls	O. Isaak	Tyndall
Tennessee	June 9-13	Memphis	F. S. Maxey	804 Bennie Dillon Bldg., Nashville 3
Texas	June 15-21‡	Dallas	R. T. Weber	312 Capital National Bank Bldg., Austin
Utah	June 23-28	Salt Lake City	M. W. Merrill	820 N. University Ave., Provo

State	Date	Place	Secretary	Address
Vermont	June 23-25	Montpelier	F. A. Reid	Windsor
Virginia	June 10-14	Richmond	J. M. Hughes	715 Medical Arts Bldg., Richmond 19
Washington	June 16-20	Seattle	J. B. Kiefer, Jr.	510 Cobb Bldg., Seattle
West Virginia	June 23-25	West Liberty	C. A. Laughlin	613 Prunty Bldg., Clarksburg
Wisconsin	June 16-20*	Milwaukee	S. F. Donovan	Tomah
Wyoming	June 23-25	Casper	W. A. Hocker	212 O & S Bldg., Casper

*Dentistry and dental hygiene.
**Dental clinical examinations.
†Dental and dental hygiene written examinations.
§Dental written examinations.
††Dental hygiene clinical examinations.
‡Dental examinations.
‡‡Hygienists' examinations.

MEETINGS OF OTHER ORGANIZATIONS

Name	Date	Place	Secy. or Chm.	Address
American Academy of Periodontology	Sept. 4-6	St. Louis	C. E. Chamberlain Secy.	115 North St., Peoria 5, Ill.
American Association of Dental Examiners	Sept. 5-6	St. Louis	C. A. Bumstead Secy.	924 Stuart Bldg., Lincoln, Neb.
American Association of Orthodontists, Central Section	Oct. 13, 14	Omaha	F. B. Lehman Secy.	1126 Merchants National Bank Bldg., Cedar Rapids, Iowa
American Board of Dental Public Health	Sept. 12, 13	St. Louis	P. E. Blackerby, Jr. Secy.	250 Champion St., Battle Creek, Mich.
American Board of Oral Pathology	Sept. 7	St. Louis	J. L. Bernier Secy.	Army Institute of Pathology, Washington, D. C.
American Board of Oral Surgery	Oct. 5-8	New Orleans	L. M. FitzGerald Secy.	718 Roshek Bldg., Dubuque, Iowa
American Board of Pedodontics	Aug. 4-7	San Francisco	R. L. Ireland Secy.	College of Dentistry, University of Nebraska, Lincoln, Neb.
American Board of Prosthodontics	Feb. 1-7, 1953	Indianapolis	I. R. Hardy Secy.	Tufts College Dental School, 136 Harrison Ave., Boston 11
American College of Dentists	Sept. 7	St. Louis	O. W. Brandhorst Secy.	4952 Maryland Ave., St. Louis 8
American Dental Assistants Association	Sept. 8-11	St. Louis	M. L. Martin Exec. Secy.	410 First National Bank Bldg., La Porte, Ind.
American Dental Society of Europe	July 15-18	London, England	F. D. Derrick Secy.	140 Park Lane, London, W. 1 England
American Denture Society	Sept. 5, 6	St. Louis	A. L. Roberts Secy.	Aurora National Bank Bldg., Aurora, Ill.
American Public Health Association	Oct. 20-24	Cleveland	R. M. Atwater Exec. Secy.	1790 Broadway, New York 19
American Society of Oral Surgeons	Oct. 8-11	New Orleans	R. F. Whalen Exec. Secy.	1112 E. Clay St., Richmond, Va.
Canadian Dental Association and British Columbia Dental Association, Golden Jubilee	June 15-18	Vancouver, B. C., Canada	C. M. Whitworth Secy.	Joint Convention Committee, Canadian Dental Association, 218 Medical-Dental Bldg., Vancouver, B. C., Canada
Chicago Dental Society	Feb. 8-11, 1953	Chicago	K. S. Richardson Exec. Secy.	30 N. Michigan Ave., Chicago 2
Eleventh International Dental Congress	July 19-26	London, England	G. H. Leatherman Hon. Secy.	35 Devonshire Place, London W. 1, England
Great Lakes Society of Oral Surgeons	Sept. 21	Cleveland	B. B. Cook Secy.	10314 Puritan Ave., Detroit 21
International Association for Dental Research	Mar. 20-22, 1953	Philadelphia	E. H. Hatton Secy.	311 E. Chicago Ave., Chicago 11
National Board of Dental Examiners	Dec. 1, 2		S. Peterson Secy.	222 E. Superior St., Chicago 11
New Orleans Dental Conference	Nov. 9-12	New Orleans	M. R. Matta Secy.	629 Maison Blanche Bldg., New Orleans
Northeastern Society of Orthodontists	Nov. 10, 11	Montreal, Canada	O. Jacobson Secy.	35 W. 81st St., New York
Odontological Society of Western Pennsylvania	Nov. 11-13	Pittsburgh	W. E. Craig Secy.	206 Jenkins Bldg., Pittsburgh 22
Southern Society of Orthodontists	Aug. 10-13	Asheville, N.C.	F. P. Bowyer Secy.	608 Medical Arts Bldg., Knoxville, Tenn.

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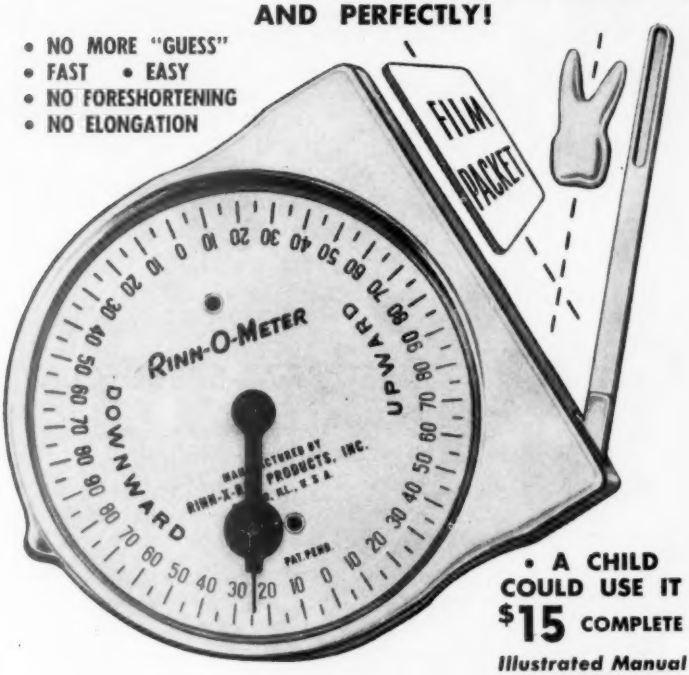

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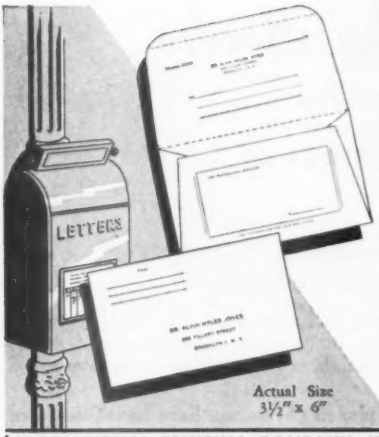

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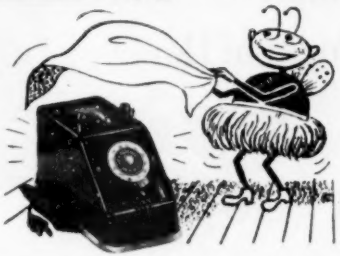

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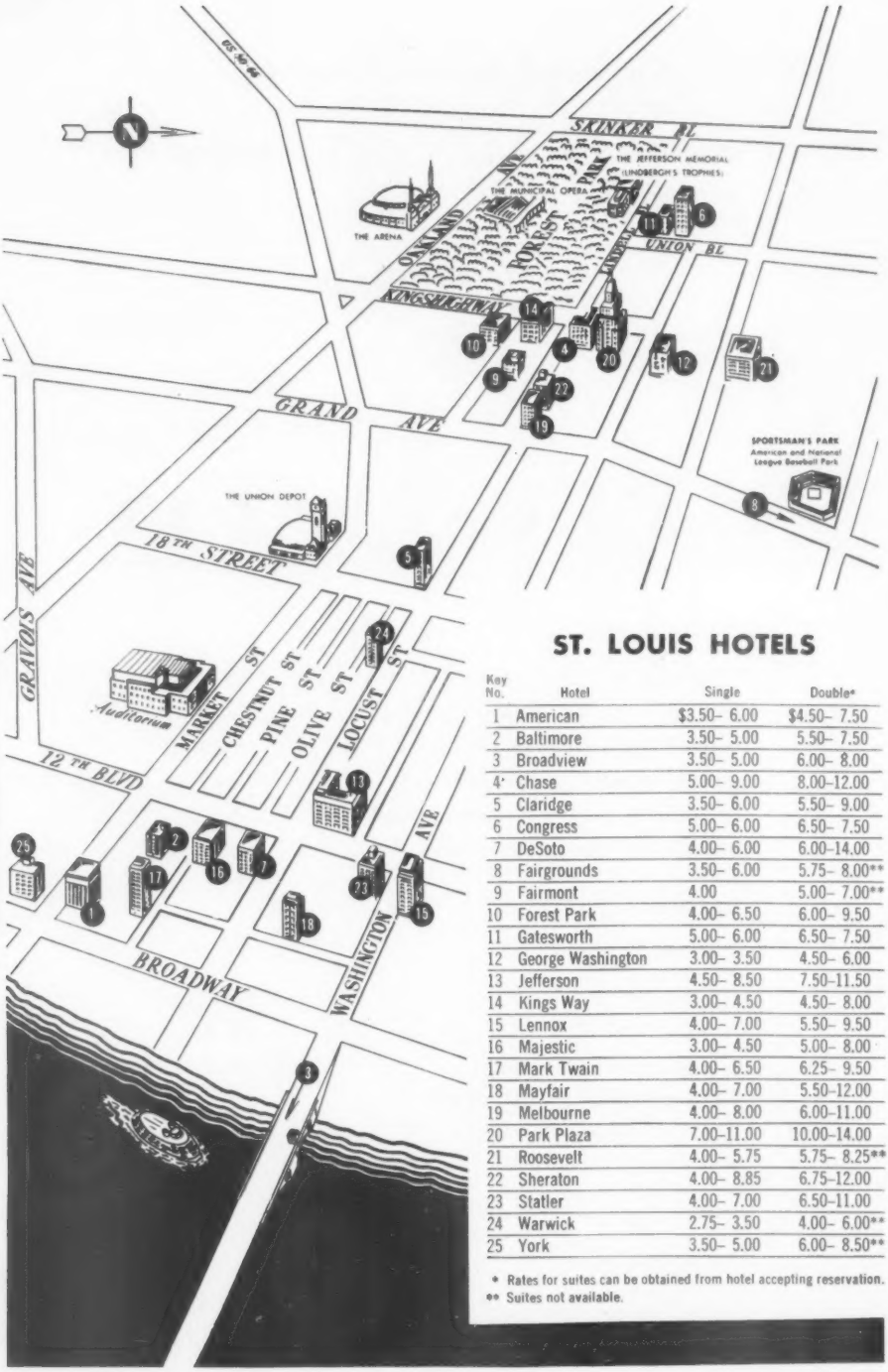

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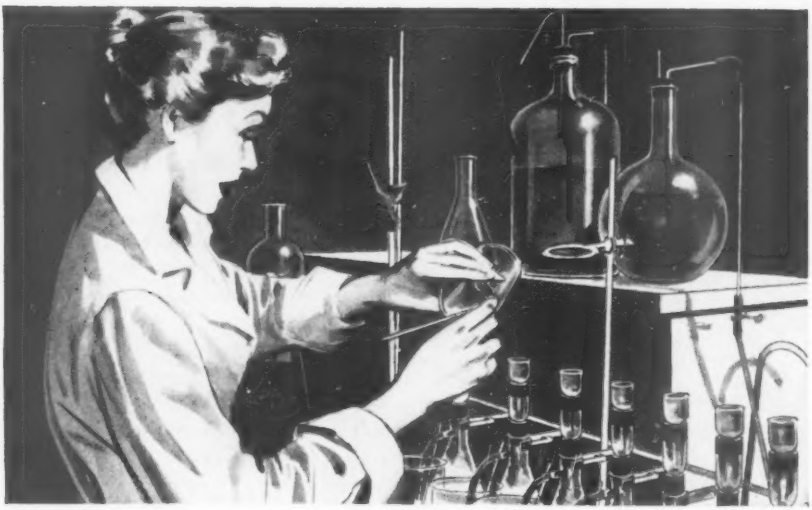

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Chemical analyses have shown ice cream to be high in nutritive value[1] . . . now research shows that certain nutrients in this favorite dairy food are more readily available to the body than are those of many other foods.[2]

A controlled diet, demonstrated to have high digestibility was fed to a group of healthy young women. After a preliminary observation period, certain foods were introduced into this basic diet in amounts in line with ordinary eating practices. Not all of these foods were well digested—failure to absorb all of the available nutrients resulted in some loss of calories, proteins and fat to the body. *No such loss of nutrients was observed when ice cream was the added food.*

The suggestion has been made that the nutritive value of certain foods may be over-estimated if the foods are judged solely on the basis of composition, with no consideration of digestibility. This is not true of ice cream which, by either criterion, ranks high among foods of nutritional importance.

1. Dahlberg, A. C. and Loosli, J. K. Nutritive value of commercial ice cream. J. Am. Diet. Assn. 24:20 (Jan.) 1948.

2. Everson, G., Pearson, E., and Matteson, R. Biological availability of certain foods as sources of riboflavin. J. Nutrition 46:45 (Jan.) 1952.

ACCEPTED COUNCIL ON FOODS AND NUTRITION AMERICAN MEDICAL ASSOCIATION

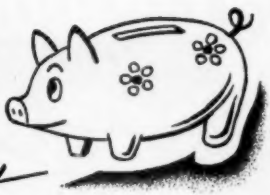
The presence of this seal indicates that all nutrition statements in this advertisement have been found acceptable by the Council on Foods and Nutrition of the American Medical Association.

National DAIRY COUNCIL

111 N. Canal Street, Chicago 6, Illinois

Since 1915 . . . the National Dairy Council, a non-profit organization, has been devoted to nutrition research and education to extend the use of dairy products.

YOU CAN BANK ON THE "PRECISION" ...WHEN YOU ORDER A *Replicast* PRECISION REMOVABLE



Why risk disappointment in the fit of a cast removable, when it costs you no more to be certain?

You'll learn to take precision fit for granted after you have inserted your first few REPLICAST removables. That's because each step in the Dresch-developed REPLICAST technic is carried out with testing-laboratory precision; the "variables" inherent in traditional casting procedures are eliminated. Yet this very fact makes for greater efficiency in production, so the cost to you is no greater—in fact, may be even less.

Decide now to enhance the scope and prestige of your partial-denture service by the regular use of these superbly-fitting removables. Mail the coupon or write for price-list and full particulars now.

REPLICAST ...A METHOD... NOT A MATERIAL

The Dresch Laboratories Company
1009 Jackson Street—Toledo 1, Ohio

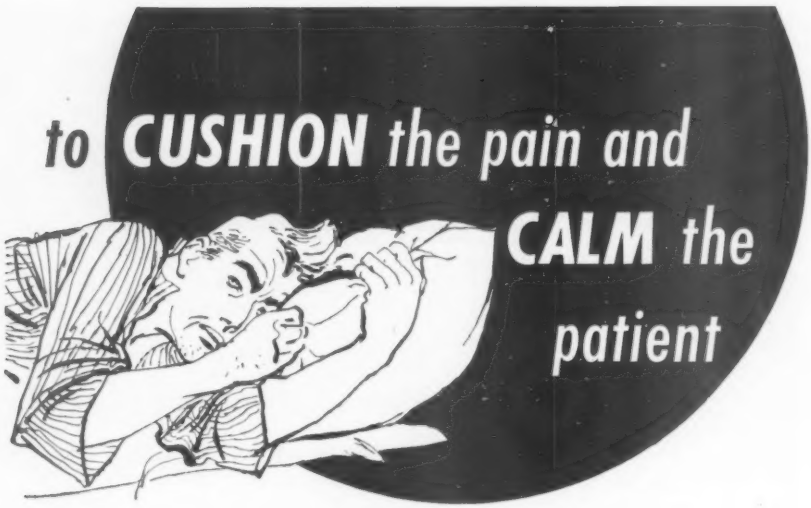
Tell me more about 'REPLICAST' precision removables.

DR._____

*Street Address*_____

*City—Zone—State*_____

J62



WHEN postoperative pain and nervousness combine to cause hours of sleepless discomfort, specify the effective relief of NEMBUTAL and ASPIRIN capsules. Here, in a single capsule, two well-known drugs work together: The 30 mg. (½ gr.) of short-acting NEMBUTAL provides enough sedation to overcome restlessness, while the 0.3 Gm. (5 grs.) of aspirin is usually effective in controlling the pain which so frequently follows extraction or instrumentation. With short-acting NEMBUTAL, onset of action is rapid and duration of effect brief. Even when more than one capsule is needed, there is little likelihood of cumulative effect, since the drug is rapidly and completely destroyed in the body. NEMBUTAL and ASPIRIN capsules are available in bottles of 100 and 500. Abbott

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(PENTOBARBITAL AND ASPIRIN, ABBOTT)

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High Purity

Unusually Close Chemical Tolerances

There are thousands of dentists who are confirmed optimists on the value of Ammoniacal Silver Nitrate in dental health. Ammoniacal Silver Nitrate has been the cornerstone of success for many dentists. They use it for what they get out of it.

You can now operate by Color.

Armed Services, to make applications of Ammoniacal Silver Nitrate *colored blue*, send for information.

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"One picture is worth more than ten thousand words." . . . If true, then Columbia Dentoforms speak volumes.

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COLUMBIA DENTOFORM CORPORATION
"The House of A Thousand Models"
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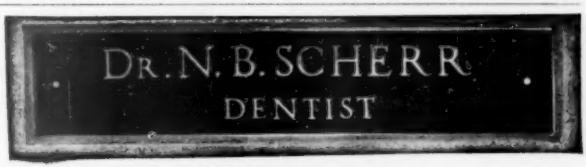

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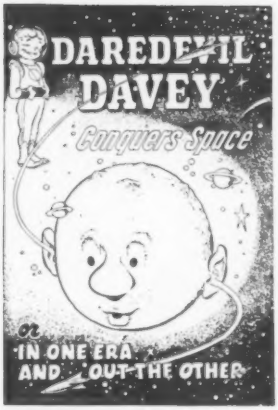

DAREDEVIL DAVEY CONQUERS SPACE

Now youngsters will doubly admire the new and even more exciting adventures of Daredevil Davey as he tells of his thrilling exploits into space while he describes in wondrous story form, the advantages of dental health. Once again Davey's dentist is right on hand between trips to help point out the important health messages given throughout the book.

Doctors, keep those young minds excitingly occupied while they are in your reception room. Parents and teachers as well as the children themselves, will appreciate copies, and you'll want several for your reception room. A free copy will be sent on request.

use this order blank

Order Department, AMERICAN DENTAL ASSOCIATION
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Check the order you wish to place for G-14, DAREDEVIL DAVEY CONQUERS SPACE booklets:

- ☐ *25 Copies . . . . . $ 2.85*
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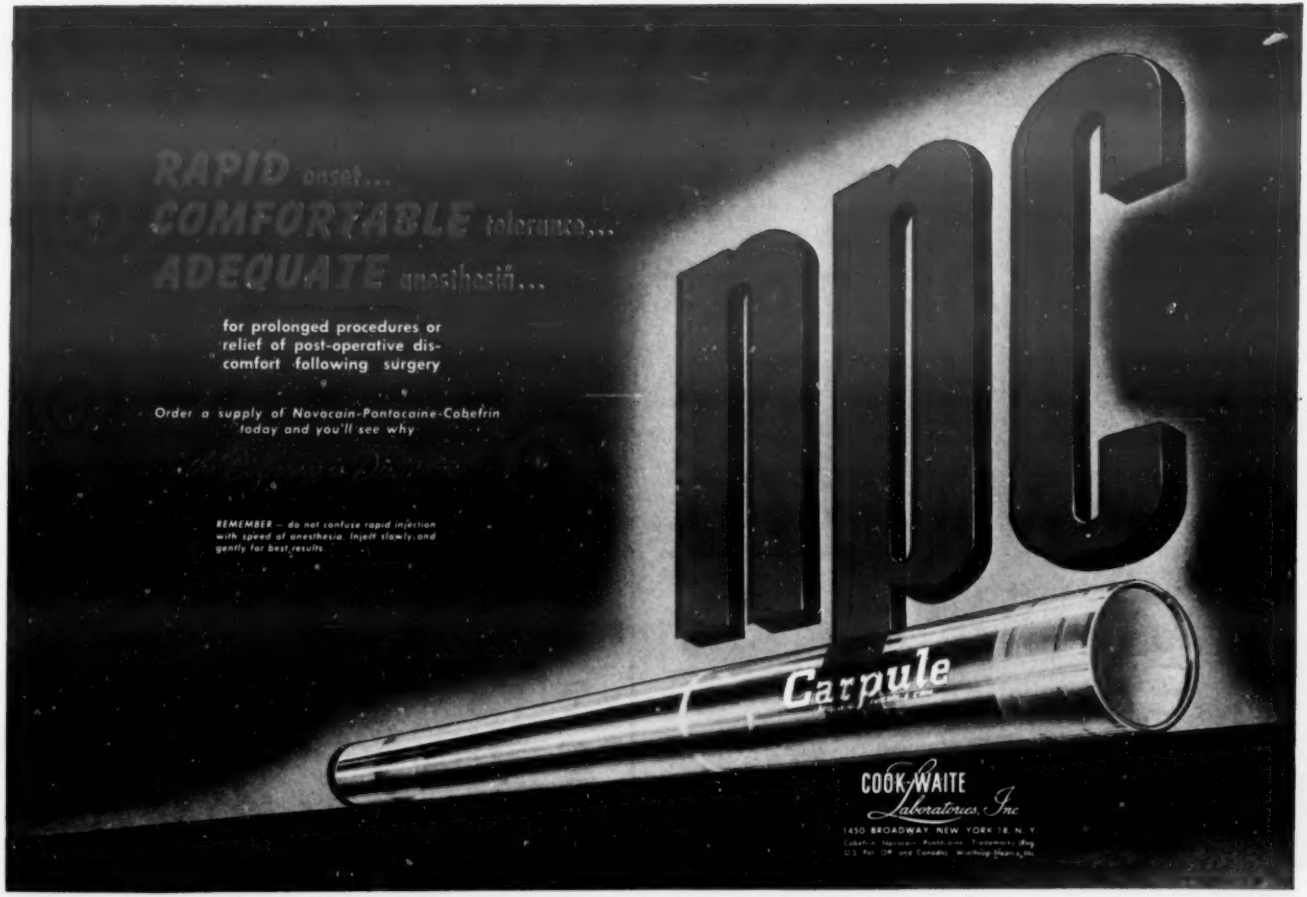
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for prolonged procedures or relief of post-operative discomfort following surgery
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REMEMBER — do not confuse rapid injection with speed of anesthesia. Inject slowly and gently for best results.
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announcing

new d-p porcelite

a low-temperature-curing denture resin

Porcelite Eliminates Dimensional Changes Caused by Processing, Produces Strong, Amazingly Accurate, Perfectly Fitting Dentures!

curing ordinary acrylic

1 Ordinary acrylic dentures must be processed at temperatures considerably above 140° F.

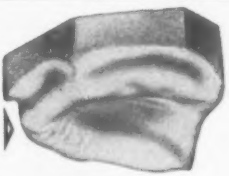
2 Above 140° F., all stone models expand.

High temperature curing with ordinary acrylics causes dimensional change and loose fitting dentures—all your pains-taking professional skill thwarted by an uncontrollable chemical reaction!

curing d-p porcelite

1 PORCELITE dentures are correctly processed at temperatures below 140° F

2 Below 140° F., with PORCELITE, models DO NOT expand.



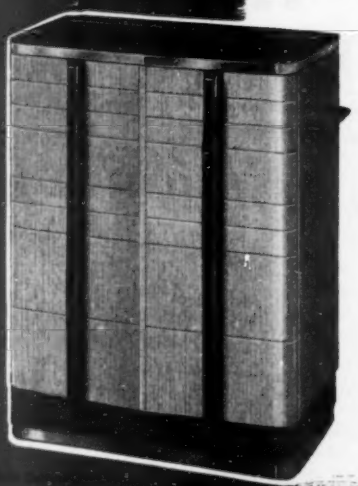
Low temperature curing with PORCELITE eliminates dimensional change and loose fitting dentures—your professional skill is rewarded by strong, completely accurate, perfectly fitting dentures!

Order from your Dental Dealer NOW!

dental perfection co.

543 West Arden Ave., Glendale 3, California

This investment has not missed a dividend



The American Cabinet Company

DIVISION OF HAMILTON [illegible] WISCONSIN

An Invitation...

IF YOU ARE attending the Congress of the Federation Dentaire Internationale which will be held in Royal Festival Hall, London, England, in July of this year, we extend to you a cordial invitation to visit the Amalgamated Dental Group's exhibit — which will occupy the center position in the exhibition hall.

We shall also be pleased to make arrangements for you to visit one of the Group's engineering plants, situated near London, to see at first hand the care, craftsmanship, and organization that insure the quality of all "Amalgamated Dental" products.

The services of the Professional and Technical Division — located at 12 Swallow Street, Piccadilly — will be at your disposal during your visit to the Congress.

11TH INTERNATIONAL DENTAL CONGRESS
Royal Festival Hall, London
July 19th-26th, 1952

Claudius Ash, Sons & Co., U.S.A., Inc.
ONE OF THE AMALGAMATED DENTAL GROUP OF COMPANIES
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Ash... the Hallmark of Quality Performance in Dental Products

ARE YOU USING THE WORLD'S FINEST ALLOY...

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ORDER THIS NEW TRIAL 11-OUNCE DISPENSER COMBINATION

The extra "Bonus Ounce" is for trial. Use it and if not completely convinced, return to your dealer for full credit.

ARISTALOY amalgam fillings stay bright and clean in the mouth—*definitely* sealing cavities, because of the great density and the special shape and size of selected Aristaloy particles.

Our newest Individual Bottles Dispenser fits directly into capsules of mechanical amalgamators. It is very easy to use and will save much of your time and money.

A few minutes of conscientious study of the directions and several trial "spills" are a worthwhile exchange for a lifetime of uniform mixes of the exact size and consistency you desire.

$28.25

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Precision Patterns Produce Precision Inlays

DuraLay PATTERN RESIN

assures perfect fit

sharper margins, faster seating

The new DuraLay method for precision patterns is simple, positive! Using DuraLay Powder and Liquid, you quickly build up pattern with brush method, register bite, remove pattern, tack on wax sprue, and cast in usual manner.

No more worry about handling delicate wax forms. No hazard of breaking while taking bite, no distortion when drawing from tooth, no lost margins.

DuraLay burns out completely. You get a smooth, accurate casting every time. You save chair time, and the annoyance of seating faulty inlays, three-quarter or full crowns.

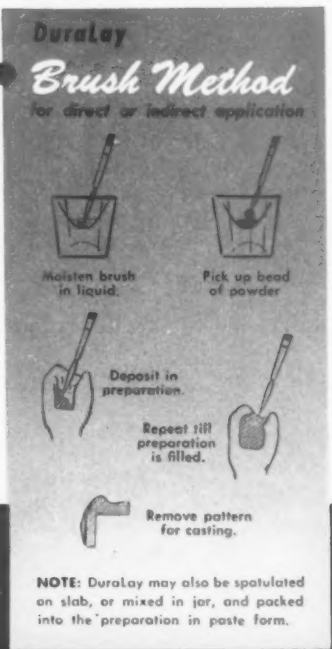
DuraLay is economical, too—a package is sufficient for approximately 200 patterns.

DuraLay is guaranteed
Use DuraLay on several patterns—if results do not fulfill your every expectation, return remainder for full credit.

Combination Kit **$8.50**
Includes Powder, Liquid, Dropper, Lubricant, Three Brushes, Two Dappen Dishes

Powder and Liquid $6.50

Order DuraLay From Your Dealer



NOTE: DuraLay may also be spatulated on slab, or mixed in jar, and packed into the preparation in paste form.

Reliance
DENTAL MFG. CO.
22 E. Van Buren St. • Chicago 5, Ill.

Exhibit number one of CDX superiority

Yes, with a CDX you get clear, precise radiographic detail . . . you complete your diagnosis while your patient is in your chair.

When considering the purchase of an x-ray unit, investigate the CDX and its principle of complete *oil immersion*. This CDX innovation permits smaller head size for easy maneuverability, efficient cooling, longer life. See your dealer or write X-Ray Department, General Electric Company, Milwaukee 14, Wisconsin. Room JJ6

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WHEN A RESINOUS FILLING MATERIAL IS INDICATED —

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Texton

Texton is ideally adapted for the pressure, brush, and inlay techniques.

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Mixes easily on a tablet or in a dappen glass
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Resists stain, especially lipstick
ph 6-7

Its 10 tooth colors and 4 modifying colors are the same as those of Filling Porcelain Improved. If you use this, your color matching technique is unchanged.

Free Technique booklet on request

THE S. S. White

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10 tooth colors
4 modifying colors
2 bottles of liquid
1 powder measure
1 request card for a free color guide
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PRICE $31.70

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6 popular tooth colors
2 bottles of liquid
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1 powder measure

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3 popular tooth colors
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1 powder measure

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How to Get

COMPLETE STERILIZATION

in LESS TIME *than Simple Boiling*

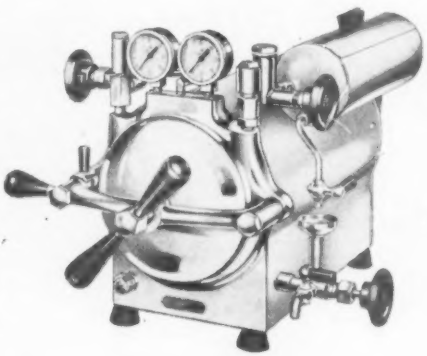
Bacteriologists and other authorities agree
1) That boiling merely sanitizes, or makes objects sanitary.
2) That only autoclaving, by utilizing steam under pressure, actually sterilizes, or completely destroys every form of life, harmful or innocuous.

And *now* that safe autoclaving, by means of the Pelton FL-2, can be accomplished in less time than simple sanitization by boiling water, why should any private office risk the danger of serious cross-infection from spore-bearing bacteria?

In addition, FL-2 autoclaving assures safe sterilization of absorbent materials, dressings and solutions. Needles, too, can be removed from the autoclave completely sterilized and perfectly dry, inside and out. Delicate instruments stay sharper, last longer, when autoclaved.

SEE the
PELTON FL-2

The FL-2 generates and then stores steam under pressure in its outer chamber ready for instant use. That means fast sterilizing. In all-day operation, current is off two-thirds of time. That means economy. One quart of water lasts one to several days without replenishing. Inner chamber is 6" x 12". Automatic controls, sturdy construction, lustrous chrome finish.



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PELTON

THE PELTON & CRANE CO., DETROIT 2, MICHIGAN

Precision Castings

EVERY TIME..IN *LESS* TIME
WITH GRAY INVESTMENT
and R & R INVESTMENT PROPORTIONER

● As every user knows, in the simplicity of its mixing and investing technic, and its ability to stand rapid burn-out, Gray Investment saves precious time in casting. If the mix is accurate, the casting will be accurate, and free from surface or subsurface pits. And now the attainment of exactly the correct powder-water mix for any type of small cast gold restoration is made quick and easy by the simple, inexpensive Investment Proportioner shown in the foreground below. By its use, the casting procedure is further simplified, and precision results more positively assured.

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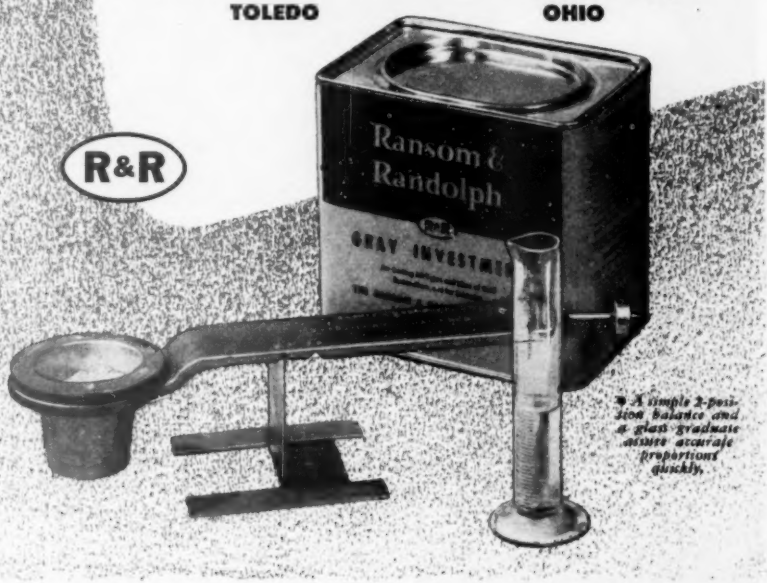

● *A simple 2-position balance and a glass graduate assure accurate proportions quickly.*

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You can prescribe Co-re-ga, with confidence in its quality and effectiveness, and in its strict adherence to the highest standards of ethical conformance. Co-re-ga (in turn) can help strengthen your new denture patients' confidence in your dentures and your professional skill, by easing the difficult period of initial adjustment.

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LOW HEAT COMPOUND

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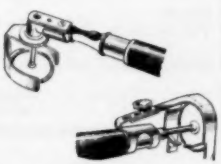

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At any age

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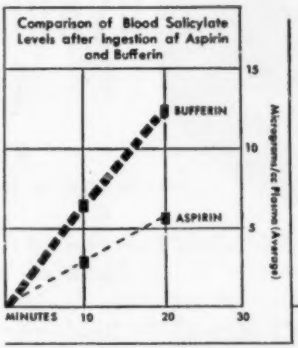
Faster Pain Relief with BUFFERIN

1 ACTS TWICE AS FAST AS ASPIRIN

The antacids in Bufferin speed its pain-relieving ingredients through the stomach and into the blood stream. Actual chemical determinations show that within ten minutes after Bufferin is ingested blood salicylate levels are higher than those attained by aspirin in twice this time.[1]



2 DOES NOT UPSET THE STOMACH

Bufferin's antacid ingredients protect the stomach against aspirin irritation. This has been clinically demonstrated on hundreds of patients.

in usual doses

In a series of 238 cases, 22 had a history of gastric distress due to aspirin but only one reported any distress after taking 2 Bufferin tablets (equivalent to 10 grains of aspirin).[3]

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In a recent study group, 1006 patients received, over a 24 hour period, 12 Bufferin tablets (equivalent to 60 grains of aspirin). Although 72 had a history of being sensitive to aspirin, only 18 reported any gastric side-effect with Bufferin.[2]

1. Effect of Buffering Agents on Absorption of Acetylsalicylic Acid. J. Am. Pharm. Assoc., Sc. Ed. 39:21, Jan. 1950
2. Gastric Tolerance for Aspirin and Buffered Aspirin. Ind. Med. 20:480, Oct. 1951

INDICATIONS: Simple headaches, neuralgias, dysmenorrhea, muscular aches and pains, discomfort of colds and minor injuries. Particularly useful when gastric hyperacidity is a complication. Useful for relieving pain in the treatment of arthritis. Helpful for toothaches and pain following tooth extraction.

EACH BUFFERIN TABLET contains 5 grains of acetylsalicylic acid, together with optimum amounts of the antacids aluminum glycinate and magnesium carbonate.

AVAILABLE in vials of 12 and 36 tablets and in bottles of 100. Tablets scored for divided dosage.

BUFFERIN is a trade-mark of the Bristol-Myers Company.
Bristol-Myers Co., 19 West 50 St., New York 20, N. Y.

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ELASTIC IMPRESSION CREAM

It's a new

HYDRO-COLLOIDAL ALGINATE

READY-MIXED

NO WATER • NO PLASTER BOWL • NO HEAT

GREATER ACCURACY. Pre-mixed at factory. Completely homogeneous. No problems of "local water chemistry" or water temperature.

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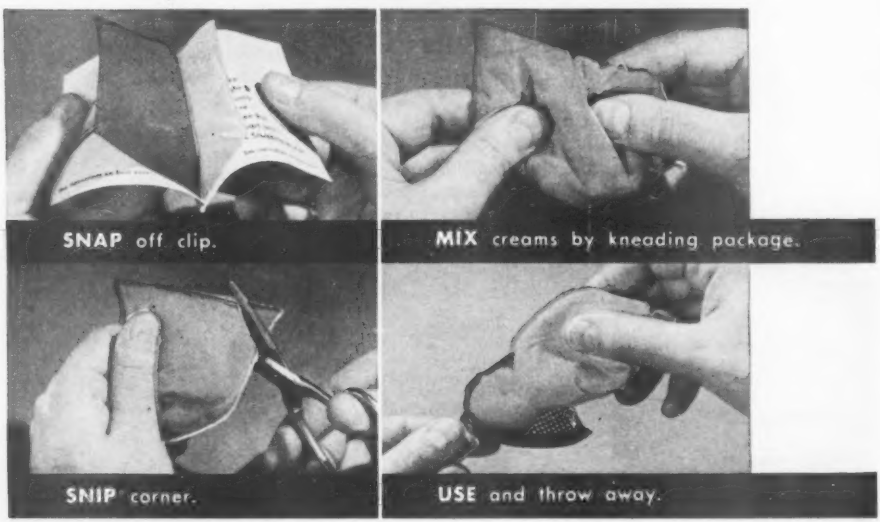

COSTS NO MORE. Call your dealer for a dozen. You'll like LS-2 better than any you've ever used. Money back guarantee.

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TRY THIS FINE FILM. Handy packages of 24 and 144, single- or double-coated, available through authorized dental supply dealers everywhere. E. I. du Pont de Nemours & Co. (Inc.), Photo Products Department D2, Wilmington 98, Delaware.

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DENTAL X-RAY FILM
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BETTER THINGS FOR BETTER LIVING
. . . THROUGH CHEMISTRY

TWO *are better than* **ONE** . . .

and three are better than two!

Such a simple "rule of thumb" in regard to x-ray equipment has proved highly advantageous for an increasing number of dentists. More and more dentists with multiple operating rooms have discovered that a Ritter DUAL-X in each room makes their practice easier . . . speeds patient examinations. More patients are handled with greater efficiency. These dentists have found, too, that each additional Ritter DUAL-X x-ray has proved to be a wise and profitable investment.

Be sure you are equipped to offer the best in dental radiography. Ask your Ritter dealer for a Ritter DUAL-X demonstration today.

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there is only one **Phospho-Soda** *(fleet)*

— safe and effective whenever laxation is indicated

Phospho-Soda (Fleet) is a solution containing in each 100 cc sodium biphosphate 48 Gm. and sodium phosphate 18 Gm. Both 'Phospho-Soda' and 'Fleet' are registered trademarks.

C. B. FLEET CO., INC.
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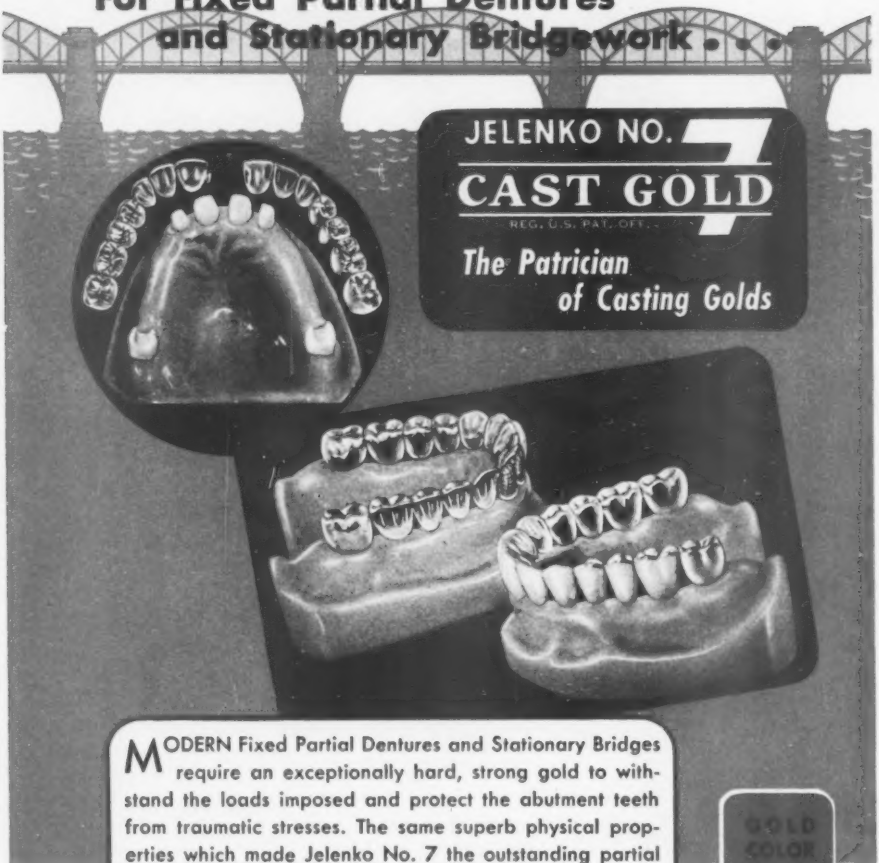
For Fixed Partial Dentures and Stationary Bridgework . . .



MODERN Fixed Partial Dentures and Stationary Bridges require an exceptionally hard, strong gold to withstand the loads imposed and protect the abutment teeth from traumatic stresses. The same superb physical properties which made Jelenko No. 7 the outstanding partial denture gold, now make it the choice of discriminating dentists for fixed partials, stationary bridges and their abutments. For long span restorations, Jelenko No. 7 is a "must."

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Cast by THERMOTROL

KADON FILLINGS

completed

efficiently

for long service

with the

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FASTER SETTING IN THE CAVITY

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Speeds the Mastery of the Denture

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JUNE 1952

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• • •

Although childhood teething has long been blamed for causing concurrent rash, cough, diarrhea and fever, modern authorities say "it ain't so"—it's just a time-sanctioned empiric belief.

• • •

History has few instances of more supreme irony than the case of Dr. C. Edmund Kells, who established the first dental X-ray clinic only one year after Roentgen's notable discovery...and ultimately died from the effect of the rays.

• • •

Dr. John Baker, dental practitioner in Boston, was apparently quite a patriot-dentist. Not only was he one of Washington's seven dentists...not only did he teach Paul Revere the art of dentistry...but his 1767 notice of setting up practice carried the first American reference to gold fillings.

• • •

About the time of the Civil War, John N. Farrar began collecting orthodontic records, which culminated in the 1888 publication of his "Treatise on the Irregularities of the Teeth and Their Correction"—first outstanding American book devoted entirely to the subject.

• • •

Gabriello Fallopio—an Italian anatomist and student of Vesalius—may perhaps be best known for his early description of the "Fallopian tubes". However, it was he who first gave an accurate report of the developmental anatomy of the teeth, in his famous "Observatio es Anatomicae," published in Venice in 1563.

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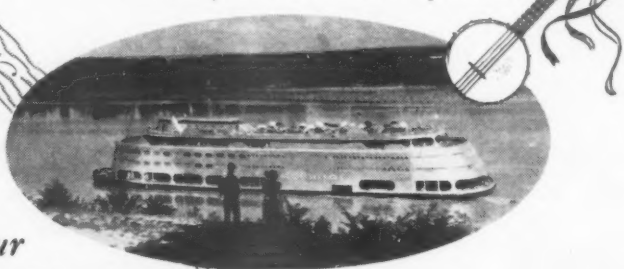

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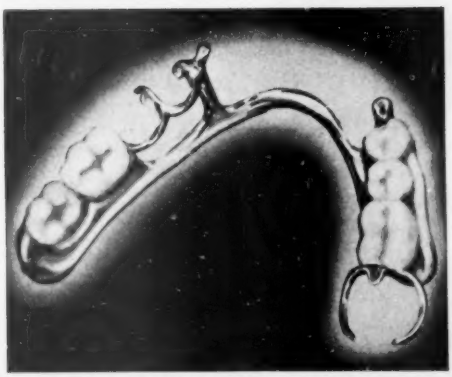

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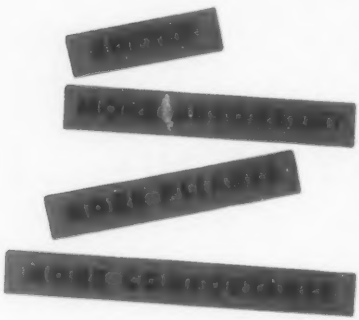

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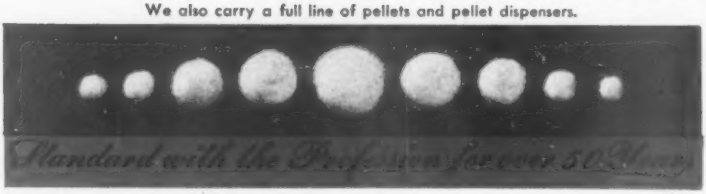

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